INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the Seventh Annual Meeting

Northwestern University Dental School, Chicago, Illinois, March 23 and 24, 1929[1]

Compiled by WILLIAM J. GIES, F.A.C.D., *Secretary*[2]

Columbia University, New York City

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I. President's Address[3]

Leroy M. S. Miner, D.M.D., M.D., Dental School, Harvard University, Boston, Mass.

Owing to the fact that a permanent secretary has been selected (at least unofficially) who has the responsibility of arranging the programs and caring for much of the ad interim business, and also to the

[1] The first and second sessions, and the fourth and fifth, were held in the Northwestern University Dental School, Chicago, Ill., where every facility for the successful conduct of the meeting was provided. The third session followed an informal dinner in the Medical and Dental Arts Building (185 North Wabash Avenue). The chairmen were Leroy M. S. Miner, Harvard University, President, and Arthur D. Black, Northwestern University, President-elect. An account of the executive proceedings will be published in the Supplement of this volume, in the number for December, 1929. Abstracts of the papers presented at the annual meeting in 1928 were published in the *Journal of Dental Research*, 1928, viii, p. 395.

[2] Most of the abstracts have been prepared by those who presented the corresponding papers. All have been verified by the authors.

[3] Delivered at the opening of the third session, after the informal dinner on March 23, and preceding the annual business meeting. See page 283.

fact that the structure of the Association is built on a number of self-governing units, it happens that the duties of the President are confined principally to presiding gracefully and effectively at the annual meeting, and to delivering an annual address. This latter duty carries a real responsibility, if such address assumes to be a declaration of principle, a guide for effective development, or an inspiration to renewed effort.

The practical idealism on which this Association is founded must ever be a source of inspiration to all pioneers on the frontiers of dental knowledge. To promote broadly the advancement of active research in all branches of dentistry, and in related phases of the arts and sciences that contribute directly to the development of dentistry, and also to encourage and facilitate coöperative effort and achievement by mutual helpfulness among investigators in all nations in every division of stomatology, to the end that dentistry may render cumulatively more perfect service to humanity, are worthy purposes that appeal both to the imagination and to the practical senses.

In the President's address last year it was pointed out that, to develop dental research most effectively, the coöperative effort of three different groups was necessary: the investigators, the administrators and the donators. The scientific program of last year's meeting and of the present meeting quite clearly indicate how well the first group is beginning to contribute to the advancement of knowledge. In order that the administrators and the donators may be able to take active part in furthering the interests of this organization, provision should be made for membership in the Association of these groups. The deans of two dental schools have expressed to me the wish that they could become members. Deans of both dental and medical schools, and directors of hospitals and infirmaries, with an interest aroused, could do much to further research in their institutions and thereby contribute to the general good. This recommendation is strongly urged.

In considering the relation of an administrator of a dental school to dental research, there arises the question of the relation of dental education in general to this problem. Dental education has been concerned chiefly with training technicians. If dental education is at all concerned with education, and not merely with vocational training, it should be concerned with the development of scientists as well as

with the training of skillful technicians. Under the most favorable conditions very few students in dentistry have the capacity for, or the interest in, both the acquirement of technical skill and of scientific achievement. Such men are exceptions at present; but when they do appear, opportunity should be provided for them to develop as broadly as possible. When a man gives evidence of being interested chiefly in the science of dentistry, in research, or in teaching, such a man should be given the largest opportunity for development.

We used to hear much about the theory courses and the practical courses. Such courses as physiology, histology, bacteriology, etc., have been regarded as subjects to be taken and passed, and perchance forgotten as soon as the so-called practical courses are under way. This has been and is a weakness in dental education. To be "practical" should not mean that science is disregarded. In fact, it should be quite the reverse. In the industrial world, the practical man promptly seizes and applies to his field the attainments of science. It is a fact that there has been a lack of progress in the prevention and control of dental ills. Is it because we have not adequately applied what science has discovered, or has there been a lack of progress in the science of dentistry itself? Have we disregarded the importance of dental research in our educational problem? The two last questions can be answered emphatically in the positive.

The criticism has been made that there is a lack of graduate work in dental education. The reason is obvious. There has been a great lack of research activity among teachers in the various fields of dentistry. This, in turn, has been due to the inability of most dental schools to pay adequate salaries to teachers. This economic problem is the most critical now facing dental education. When this is solved, men will be available. Here is a large opportunity for the donators. The recent gifts to Yale and Rochester, for the development of dental research on a broad basis, is indicative that an appreciation of the situation exists. There is then reason for optimism. This meeting radiates it.

Here and there men endowed with a thirst for new knowledge—who have always the inquiring mind—are beginning to produce interesting and valuable results. Along this route progress will be made. Research is a matter that cannot be standardized or organized too highly to advantage. Each problem is an adventure into the frontiers of

knowledge. Vision, imagination, courage, patience, and persistence are as important and necessary as acquaintance with the field of knowledge already conquered, and with the fundamental principles involved in acquiring that mastery. But research may be nurtured, developed, stimulated, encouraged, so that the spirit of inquiry may become ablaze with new energy. To nurture, to develop, to stimulate, to encourage must ever be important functions of this Association.

II. First Session: Morning, March 23

1. Bone Regeneration Following Extraction of Teeth in Dogs

Edward H. Hatton, B.L., M.D., and Warren R. Schram, D.D.S., Northwestern University Dental School, Chicago, Ill.

One of us has previously discussed the healing of the sockets of teeth in dogs after extraction, by the forceps and also by the so-called open-view or surgical method. In the former procedure this concerns the rapid organization of a blood clot by vascularization, and its conversion into very young connective tissue. The cribriform plate of the socket disappears very early—within one to two weeks. The adjacent new connective tissue becomes very richly cellular. These cells arrange themselves in the form of columns projecting perpendicularly from the surface of the socket, and within the spaces so made bone is formed. This process spreads rapidly into the center of the socket until the whole is converted into spongy bone. In the second case, so-called surgical removal, the method is essentially the same. But since the blood clot is quite scanty, and the muco-periosteal flap is sutured across the space formerly occupied by the tooth, the socket is nearly obliterated. Roughly speaking, it may be said that a central, bucco-lingual section of the new bone is rectangular in shape. But in the forceps extraction, a similar longitudinal section of the surgical extraction is a right angled triangle. In other words, in the end result there is less bone in the surgical removal than in the forceps extraction. This variation seems to be due to the limitation placed upon regeneration by the position of the muco-periosteal flap.

This same material seems to present evidence as to the source of new bone and the ability of the periosteum to function as an osteogenetic layer. In performing the surgical operation, the periosteum

was carefully stripped from the bone so as to preserve it intact as far as possible. The buccal plate was then chiseled away, the tooth tipped out of the socket, and the flap sutured into place. In spite of this, in none of the sections made from five sockets at various stages of healing has bone been detected proliferating from the periosteal flap. In every instance bone regeneration has begun at the margins of the bone bordering the former socket, and spread from this zone toward the periphery until it came adjacent to the old periosteal layer, where bone formation ceased. The fact that bone formation ceases at this point suggests that the periosteum is a limiting plane of tissue, which, by its position, controls the extent to which new bone is produced. The pressure of the periosteal flap on the old crest of the alveolar lingual bone plate, and at the lower angle of the wound on the buccal plate, seems to cause resorption of the bone in both of these locations. On this account, not only is less bone produced in this type of regeneration, but an additional portion of the original bone is removed by a resorbing process.

These facts support the opinions of a very considerable group of investigators who contend that periosteum is not a truly osteogenetic tissue, but that its chief function is to control and limit bone formation as well as to furnish the mechanism for the attachment of the soft tissues. It may be contended that, in this operation, the osteogenetic layer of the periosteum was not stripped from the buccal plate and that it was entirely lost with the bone. On the other hand, it is more likely that in places all of the periosteal layers were included in the flap. Since no regions of bone regeneration have been observed to originate from the periosteal flap, we conclude that, in this instance, osteogenesis is not a function of the periosteum.

2. THE DENTAL ARCHES OF IDENTICAL TWINS

Samuel Goldberg, M.S., L.D.S., D.D.S., Northwestern University Dental School, Chicago, Ill.[4]

It was the writer's intention merely to use this valuable material for the information it might yield on a number of dental problems.

[4] The author is greatly indebted to Prof. H. H. Newman, of the University of Chicago, for advice and assistance in the conduct of this investigation and for the opportunity to study the mouths of twins in the clinic there. A more complete statement of some of the data is published on succeeding pages: *Journal of Dental Research*, 1929, ix, p. 363.

But the value of the dentition from a general biological point of view soon became obvious, and equally important, inasmuch as it afforded additional evidence on the hypotheses of Prof. H. H. Newman of the University of Chicago in the study of several other characters.

By comparing the left and right sides of one twin with those of the other it is possible to determine whether the asymmetry mechanism has operated in any given case to produce differences other than those imposed by environment. Of 42 pairs of twins examined by the writer, 10 bore good evidence of reversed asymmetry. This evidence has to do with such conditions as reversals of typical deformities and of the side preferred for chewing. In 50 pairs of identical twins there were 42 cases of malocclusion. Of the 42, only 14 were due to extrinsic factors in development. Even some of the 14 are doubtful; a just estimate would not exceed 10. Every type of malocclusion was represented on an inheritance basis. Except in individual cases, where abnormal habits interfered with intrinsic forces, the arch form, as previsioned in the germ cells, is carried to fruition in the adult, and there is very little justification to assume that environment, either intra-oral or extra-oral, under the average conditions in life, plays a very great or very frequent rôle.

The side preferred for mastication is closely related with handedness. Since handedness is inherited, it was concluded that the side preference for chewing is also inherited. The consensus of authoritative opinion is against changing handedness. It is concluded, therefore, that the advice expressed in the slogan, "chew on both sides," is at least somewhat superfluous.

There is no evidence to suggest that heredity plays any part in the incidence of decay occurring or commencing on the smooth surfaces. On the other hand, decay in pits and fissures in a number of twins was rather uniform, suggesting that the heredity mechanism, by determining the position of pits and fissures, may affect the location of caries.

3. THE FLUORESCENCE OF TEETH AS ANOTHER METHOD OF ATTACK ON THE PROBLEM OF DENTAL CARIES

H. C. Benedict, A.B., M.A., Chemical Department, Northwestern University Dental School, Chicago, Ill.

Under the exciting influence of ultra-violet rays, the dentine of teeth fluoresces more brightly than the enamel. A beginning-carious

lesion, or a similar lesion produced by dilute acetic acid, does not fluoresce, while decalcified dentine retains this ability. An ashed tooth does not fluoresce, nor does dentine boiled in strong sodium hydroxide. This seems to indicate that it is the organic matter in the tooth which fluoresces. A further study of the fluorescence of the materials in teeth may indicate the steps in the production of a carious lesion.

4. A CASE OF COMPOSITE ODONTOMA

Herbert A. Potts, D.D.S., M.D., Department of Oral Surgery, Northwestern University Dental School, Chicago, Ill.

This tumor was taken from the mandible of a three-year old child because of its tendency to increase in size, and because of the suspicion that it might be malignant. The mass is oval, having diameters of about two and three centimeters, and consists of both soft and hard tissues in about the proportions of three to one. The soft structures are made up of an embryonic connective tissue enclosing strands, cords, and nodules of tooth-follicle tissues, including both enamel-forming cells and odontoblasts. Both enamel and dentine are present and arranged in a very haphazard way. Only in a few instances is there any semblance to tooth form. No cementum was found in any of the numerous sections studied. The specimen is of particular interest because of the youth of the patient, which probably accounts for the complete absence of the cementum and other peculiarities of the tumor.

5. A STUDY OF THE REPLANTED TOOTH IN THE DOG

William G. Skillen, D.D.S., and G.R. Lundquist, D.D.S., Northwestern University Dental School, Chicago, Ill.

The basis of this study was not so much the replanted tooth as such, as the biology of the dental tissues as a whole. The present report deals with preliminary experiments that have paved the way for a more exact technique, and which cover periods of one month and four months respectively. No final conclusions may be drawn at present.

Reattachment of the untreated tooth occurred through formation of cementum, the periodental membrane usually assuming a normal arrangement and appearance. The treated tooth suffered much more extensively through resorption than the untreated. Fusion of bone

with cementum also occurred in the former. This might be accounted for by the slight difference in handling. Pulp and peridental membrane reunited in the untreated tooth. In a month, however, the pulp exhibited atrophy accompanied by an osteoid-like tissue formation on the walls of the canals. The gingival epithelium, under favorable circumstances, seemed to have returned to normal, although no evidence of its reattachment to enamel could be obtained. No changes of any sort occurred in the hard tissues in regions of acute inflammation.

6. EPITHELIUM IN THE PULP

J. R. Schumaker, D.D.S., Northwestern University Dental School, Chicago, Ill.

Römer and Mueller have observed epithelium in the pulps of teeth with granulomas present. The author presented three similar cases, in which granulomas were also exhibited. It was apparent that the epithelium in the pulp originated in the apical region. An instance of a secondary cuticule, on a resorbing deciduous tooth, was also shown.

7. BACTERIOLOGY OF VITAL TEETH

Anna C. Nichols, B.S., M.S., Northwestern University Dental School, Chicago, Ill.

The bacterial findings for nineteen vital teeth, cultured under both aerobic and anaerobic conditions, were given in this preliminary report. The teeth were free from decay, although a slight pyorrheal condition was present. Microörganisms were found in 41 per cent of vital pulps. Streptococcus was the predominating organism among those isolated. In a large majority of cases, it grew only under anaerobic conditions.

III. SECOND SESSION: AFTERNOON, MARCH 23

8. THE DENTO-CEMENTAL JUNCTION AT THE APEX OF A TOOTH

Edgar D. Coolidge, B.S., D.D.S., Chicago College of Dental Surgery, Loyola University, Chicago, Ill.

Having in mind the anatomical relation of the cementum to the dentin, in the apical region of the dog's teeth, it has been a general belief that the root-canal is constricted at the apical foramen. This constriction has often been spoken of as the dento-cemental junction.

The author presented a number of slides from human material of different types of teeth at different ages. In these pictures it was shown that the dento-cemental junction at the apex is not a junction, a line or a point that would have any practical bearing upon the removal of the pulp or a filling of a root-canal. The apical opening, instead of being constricted, is sometimes straight, sometimes divergent, and often on the side of the root some distance from the apex.

9. CONTRIBUTION TO THE KNOWLEDGE OF THE DEVELOPMENT OF THE DENTIN

B. Orban, M.D., Chicago College of Dental Surgery, Loyola University, Chicago, Ill.

On specially treated specimens, embryologic as well as adult, it has been shown that the dentin matrix is produced by the transformation of argyrophile fibers of the pulp into collagenous fibers. The odontoblasts seem to have the function of nourishing the dentin and also probably of calcifying it.

10. RETROGRADE CHANGES IN PULPS OF TEETH, WITH REFERENCE TO TREATMENT

Edgar D. Coolidge, B.S., D.D.S., Northwestern University Dental School, Chicago, Ill.

Pulp conditions were shown where there were extensive calcifications of the pulp tissue in the apical region. The relation of these mechanical obstructions to the treatment of the canal was discussed. It was concluded that, if such structures were not infected or could be disinfected by suitable treatment, satisfactory results might be accomplished even though such obstructions could not be entirely removed during the treatment.

11. THE DEVELOPMENT OF THE BIFURCATION OF MULTI-ROOTED TEETH

B. Orban, M.D., Chicago College of Dental Surgery, Loyola University, and Emil Mueller, M.S., D.D.S., Northwestern University Dental School, Chicago, Ill.

The authors investigated the development of the bifurcation by means of serial sections through rat molars. They found that the bifurcation is determined long before the crown development is completed. The roots are first outlined by eccentric peripheral enlarge-

ment of the basal opening of the tooth germ. In this eccentric growth, certain portions of Hertwig's epithelial sheath remain relatively fixed, though later these projections enlarge and unite to form the bifurcation that demarcates the roots.

12. A CASE OF PERSISTENT CANINE IN A DOG

William A. Figg, Senior student, Chicago College of Dental Surgery, Loyola University, Chicago, Ill.

Illustrating with lantern slides, the author showed a case of persistent deciduous canine in position with a permanent tooth. The root of the deciduous tooth showed extensive resorptions. At one place in the gingival area, the surface of the deciduous tooth was grown together with the alveolar bone. The bone-marrow spaces and the periodontal membrane were in wide communication, and both had undergone marked fibrous changes in structure. These modifications may find their explanation in the altered function of the tooth.

13. MORPHOLOGICAL FEATURES OF THE ALVEOLAR PROCESS

Kaethe W. Dewey, M.D., School of Dentistry, University of Pittsburgh, Pittsburgh, Pa.

Because of the constant remodeling of the alveolar process, owing to the movements of the teeth, this bony structure throughout life retains some of the characteristics of young growing bone. Multiplicity in form and general appearance is a striking feature of the periosteal as well as the endosteal bone, the variations arising chiefly from morphological differences in the fibrillar constituents of the matrix, including Sharpey's fibers. Sections stained by Bielschowsky's method reveal a very striking similarity, as to texture and density, between periosteal bone and the adjacent connective tissue, and an equally striking contrast between endosteal bone and the soft tissue of the enclosed spaces. The great similarity between periosteal bone and the superimposed connective tissue strongly supports the view that such bone develops by metaplasia. The so-called bundle bone of the alveolar process is periosteal bone, with an abundance of Sharpey's fibers, and genetically identical with the coarsely fibered trellis-bone of fetal life and the so-called fiber bone at the place of insertion of tendons and ligaments in the adult. Sharpey's fibers are not perforating, but are passively included, as G. V. Black pointed out in 1887. Straight

thick bundles from the periodontium may pass directly into the bone, but relaxed interwoven periodontal fibers may also be collected and twisted into thick rope-like bundles near the alveolar wall by lodgement of columns of densely set osteoblasts. The thickness and length of such bundles in the bone is therefore also accidental, and does not represent any particular strengthening of the attachment, since the shallow implantation of Sharpey's bundles into the thin layer of young cementum apparently assures as firm an attachment.

Sharpey's fibers occur in endosteal bone, where they are always slender, since the connective tissue in the medullary spaces and haversian canals is delicate. Endosteal bone is always described as lamellar bone. However, a non-lamellar type exists. This occurs in larger and smaller masses of long fibers running parallel to the surface in which it is laid down, or irregularly in a longitudinal direction. The fibers may parallel one another, or spread fan-like, or be braided around the bone cells. This type of bone is observed chiefly in the alveolar process of young, growing individuals. As the bone becomes more mature, it diminishes, and is crowded out by lamellar bone. Such bone was described by M. Meyer, in 1927, as a peculiarity of the adult pre-cartilaginous bony capsule of the labyrinth of the ear. Mention of such bone has been made in some works on bone, but only with reference to its occurrence in enchondral bone in the earliest periods of development, and always as being laid down on remnants of cartilage It has never been mentioned as occurring in the alveolar process, where however it is common, and moreover entirely independent of pre-cartilaginous bone formation.

14. MECHANISM OF INTOXICATION BY INTESTINAL ABSORPTION IN CHRONIC ORAL SEPTIC FOCI[5]

Lloyd Arnold, M.D., and C. W. Stuart, D.D.S., University of Illinois, Colleges of Medicine and Dentistry, and the State Department of Public Health, Chicago, Ill.

The changes in the intestinal bacterial flora in oral sepsis can be best explained by the systemic intoxication that accompanies such lesions. The swallowed septic material plays a minor rôle in this condition. The lumen of the upper part of the small intestine is relatively free from bacteria in the normal individual. This segment

[5] Published in full on succeeding pages: *Journal of Dental Research*, 1929, ix, p. 305.

of the alimentary tract is slightly acid in reaction. Chronic septic foci cause changes in the parenchyma of various organs of the body. Faber has described those taking place in the stomach. The hypochlorhydria, or even achylia, causes a loss of the usual acid radicals that are necessary components of the contents of the gastro-intestinal tract in the maintenance of the normal acid-base equilibrium. The resulting tendency toward an alkaline reaction is accompanied by a loss of the self-disinfecting power of the upper half of the small intestine. Under these conditions the fecal bacteria migrate upwards from the large intestine and the ingested bacteria are not destroyed. This has led to the assumption that septic swallowed material passes the gastric barrier by reason of its own toxicity, acting locally upon the wall of the stomach. Large amounts of bacteria are readily destroyed when placed in the duodenum in the normal animal. This auto-sterilization mechanism is destroyed when the reaction is changed to a neutral or an alkaline one.

Calcium salts are soluble in an acid medium. When the gastric mucosa is changed, owing to systemic intoxication by absorbed material from a chronic septic focus, the stomach contents is not properly acidified, the upper intestinal contents becomes alkaline, and inorganic-salt metabolism is interfered with to a great extent. An interference with calcium absorption starts a vicious cycle that augments the calcium loss from the septic foci in the oral cavity.

Experimental evidence was shown in support of the idea that bacteria and bacterial products can be absorbed from the duodenum when the contents are made alkaline in reaction.

Intoxication by absorption from the intestinal tract in chronic oral sepsis is due to changes in the self-disinfecting power of the contents of this tract, and also to alterations in metabolism that are brought about by changes in the permeability of the duodenum and upper jejunum.

15. INTESTINAL ABSORPTION OF B. COLI TOXINS PRODUCTIVE OF CHRONIC COLITIS[6]

C. W. Stuart, D.D.S., University of Illinois, College of Medicine, Chicago, Ill.

B. coli produces two types of toxins: one within twenty-four to thirty-six hours in broth cultures; the other, in old broth cultures, in

[6] See the preceding abstract and footnote 5.

fourteen to twenty-one days. Berkefeld filtrates of young B. coli cultures induce convulsions and death when injected into rabbits. Filtrate from old cultures, when large doses are injected into rabbits, causes diarrhea and finally death. The neuro-tropic toxin is destroyed at 75°C., the entero-tropic toxin is heat stable. The endotoxin (entero-tropic) produces chronic colitis in a rabbit when injected in small doses during a prolonged period, the same results ensuing after administration by mouth. Two cc. of this sterile endotoxin filtrate, given to a rabbit by stomach tube every other day for twenty days, induces chronic colitis, loss of weight and appetite; and, if continued, causes death.

When the contents of the alimentary tract become alkaline in reaction, B. coli ascends from the large intestine, and becomes a part of the bacterial flora of the small intestine, up to the pylorus. This change in reaction takes place in chronic oral sepsis, as explained in the preceding paper (14). Abnormal presence of a rich B. coli flora, in the upper levels of the alimentary tract, which are normally acid in reaction, is associated with the absorption of disintegrated products of B. coli. In animal experiments, this condition causes chronic colitis. When small doses are given by mouth over a long period, there is anemia, loss of appetite, and loss of weight. Intravenous or per os administration produces the same results. The latter, then, must act by absorption through the small intestine, and thence by way of the hematogenous route to the large intestine.

16. PROBLEMS IN TEACHING PRE-CLINICAL SUBJECTS, WITH SPECIAL REFERENCE TO THE PREPARATION OF THE STUDENT FOR RESEARCH[7]

Isaac Schour, B.S., D.D.S., M.S., Department of Histology, College of Dentistry, University of Illinois, Chicago, Ill.

The problems involved in the teaching of pre-clinical subjects are essentially the same for both medical and dental students. There is a difference only in the choice of clinical application and of illustrative material, and in the emphasis on phases of special interest to the dental or medical profession. Dental students, early in their training, should become acquainted with the importance of the coöperative relation-

[7] To be published in an early issue of the *Journal of Dental Research.*

ship between the medical and dental professions. There is no better time to emphasize this than when teaching the pre-clinical subjects, which, although by tradition called "medical," are just as much *dental* as medical.

One of the common problems in pre-clinical teaching is correlation, which is of paramount importance. The responsibility for correlating all the subjects in a given curriculum must be shared equally among all of the departments. Nothing can be a greater incentive to the student's interest and concentration than the association of a given theoretical fact with a given clinical practice or experience. The application of the Golden Rule would solve many problems that come up between teacher and student. The teacher must help the student to find himself in his work, and above all encourage him to develop spontaneous interest in the subject. The teacher should guide the student's differentiation among the necessary, useful, and ornamental facts.

There are two schools of thought in respect to methods of teaching. The methodic school believes in giving a systematic and comprehensive course concentrated on fundamental facts and relationships. This method is best suited for the teaching of science to beginners. The research method employs the seminar type of instruction, and is ideal for graduate or elective courses. It is ineffective when employed in the teaching of sciences to beginners. We cannot expect students to appreciate and understand research before we help them get the A-B-C and grammar of science. The lecture has been regarded as an antiquated method, but it still holds an important place in teaching. Throughout the various biologic sciences there reign certain principles which, when understood, help to clarify the facts and integrate the parts into one harmonious whole. An introduction to these principles must be supplied in the form of lectures.

Research involves a consummation of qualities and attitudes that transcend the usual methods of teaching. A number of methods may be enumerated that help to prepare the student for research. But more important than any or all of these methods is the cultivation of an atmosphere of scientific inquiry that should permeate the lecture halls, the laboratories, and the clinics. Hunger for knowledge, passion for truth, keen power of observation, infinite patience, are among

the qualities essential to research. These qualities must come from within and must be cultivated, throughout the entire college career, side by side with the teaching of the prescribed courses. Rather than "crowd" a course and stifle the student's opportunity to search for himself, it is better to omit certain phases of the subject, and thus free some of the time for slow, careful, and spontaneous observation and reflection.

The responsibility for instilling in students an appreciation of, and a desire for, research rests on all departments and on every member of the faculty. No one department can have a monopoly of research. We know that only a very small percentage will actually do research. But it is equally important that we inculcate in the minds of all students an appreciation of, and respect for, research so that they may respond to the echoes that come from the research laboratory, and so that they may eagerly await the findings of research and help to test their application.

IV. Third Session: Evening, March 23

17. President's Address

Leroy M. S. Miner, D.M.D., M.D., Dental School, Harvard University, Boston, Mass.

Published in full on pages 269–272. The address was followed by the annual business meeting.

V. Fourth Session: Morning, March 24

18. The Relative Antibacterial Action of Various Solutions Advocated in the Treatment of Periapical Infection by Electro-Sterilization

Louis Grossman, D.D.S., D.M.D. (Rostock), and J. L. T. Appleton, Jr., B.S., D.D.S., School of Dentistry, University of Pennsylvania, Philadelphia, Pa.

The antibacterial effect of several solutions, advocated for use in the treatment of periapical infection by "electrosterilization," was determined *in vitro*. Oral bacteria and clinically tolerated dosages of current (30 milliampere minutes) were used. The conclusions on the next page were drawn from the results obtained:

(1) Greater antibacterial effect was uniformly obtained from a chemical agent that was electrolysed than from the same agent without electrolysis.

(2) Of the chemical agents tested, the one showing the greatest antibacterial effect with electrolysis was the one designated as "Solution no. 3." This consisted of zinc iodid, two parts; iodin crystals, four parts; and distilled water, 100 parts—all by weight. This was suggested and prepared by Dr. Hermann Prinz.

(3) The antibacterial effect was always distinctly greater at the positive pole. This was equally true when an iodid-iodin solution was used.

(4) The zone of sterility obtained was not due to a change in the degree of acidity of the culture medium caused by the electrolytic process.

(5) The irritant action of "Solution no. 3," when electrolysed, on subcutaneous connective tissue, does not exceed that of Lugol's solution or of physiologic sodium chlorid solution under the same conditions.

19. BLOOD COUNTS IN TWENTY-TWO CASES OF VINCENT'S STOMATITIS

Carrie Kirk Bryant, B.S., School of Dentistry, University of Pennsylvania, Philadelphia, Pa.

The cases varied in age from seventeen to thirty-three years. Eleven were college students. Sixteen were males, six were females. Thirty-six per cent showed a slight decrease in the hemoglobin, and 36 per cent showed a slight decrease in the number of red blood cells. Only one case, or 4.5 per cent, showed a slight leukocytosis. Thirty-six per cent showed a decrease in the polymorphonuclears, which were below 47 per cent in 13 per cent of the cases. The lymphocytic percentage was increased in 50 per cent of the cases.

In four cases a second count was made after clinical improvement had been brought about by treatment. In two of these cases the first count showed a marked increase in the lymphocytic percentage, with a decrease in the polymorphonuclear percentage. The second count in both cases showed a tendency to return to normal, as is shown in the summary on the next page:

		Polymorphonuclear percentage	*Lymphocytic percentage*
Case 4	Before treatment	42.0	53.0
	After treatment	68.0	20.0
Case 5	Before treatment	37.5	51.0
	After treatment	65.5	26.0

20. BLOOD TYPES IN PYORRHEA ALVEOLARIS

K. Polevitzky, M.D., School of Dentistry, University of Pennsylvania, Philadelphia, Pa.

This study was undertaken to learn whether the distribution of blood types affords any evidence for or against the inheritance of a predisposition to pyorrhea alveolaris. Blood types are known to be inherited. There is considerable evidence that their inheritance is correlated with certain pathological states. Weber and Pastern[8] found a slight increase in Group A (N. R. C. classification) among pyorrhea cases, as shown by the following summary of their data:

Group	*Normal per cent*	*Pyorrhea per cent*
O	42.0	32.5
A	44.6	56.1
B	11.0	8.1
AB	2.5	3.3

Their findings, based on 369 cases, were in general confirmed by my own examination of 100 cases, the results of which are summarized below:

Group	*Normal per cent*	*Pyorrhea per cent*
O	42–45	41
A	40–41.4	49
B	10–10.4	6
AB	5– 5.4	4

21. ON THE GROWTH OF THE MOLAR TEETH AFTER ERUPTION IN THE ALBINO RAT AND IN THE CAT

H. H. Donaldson, Ph.D., Sc.D., Wistar Institute of Anatomy, Philadelphia, Pa.

In the albino rat the maximum increase from puberty to maturity in the average length (mesio-distal diameter) of the maxillary row of

[8] Weber and Pastern: *Deutsch. Monatschr. f. Zahnheilkunde*, 1927, xlv, p. 704.

molars is 10 per cent for the male; 9.4 per cent for the female. In the case of the mandibular row of molars, the figures are 9.5 per cent for the male; 10.7 per cent for the female. In both sexes the transverse diameters (labio-palatal) also increase with advancing age. In the maxillary rows, the increase is 10.6 per cent for the males; 10.3 per cent for the females. In the mandibular row, 10.3 per cent for the males; 7.8 per cent for the females. These observations were made in collaboration with Dr. Harley E. French.

If these differences were attributable to the greater chance of survival of the larger animals, with correspondingly larger teeth, to the higher age series, one would expect to find some rows in the younger group giving measurements as great as those in the older group. This however is not found. In no case in the young animals does the row with *greatest* length equal the row with the *least* length in the old group. Consequently, the only way the rows of teeth represented in the young group can attain the length of those in the old group is by growing larger with increasing age and body size.

Measurements on three series of cat skulls gave similar results. In each case the larger skulls have larger teeth, and the larger skulls are those from older animals. Further, the highest values in the *young* groups are, with one exception, less than the lowest values in the *old* groups.

The rat representing the rodents, and the cat representing the carnivores, both show this increase in the size of teeth with age. The data presented here afford additional evidence against accepting unquestioningly the general belief that the enamel of erupted teeth is an inert, unchangeable substance.

DISCUSSION[9]

Theodore B. Beust, D.D.S., M.D., Dental School, University of Louisville, Louisville, Ky. I have some contributory evidence to add to the observations of Dr. Donaldson. During the year preceding the 1927 meeting of the American Dental Association at Detroit (October, 1927), while engaged in the study of enamel development in the pig's mandible, differences in size of the teeth in old and young jaws were evident. This was most

[9] Discussions have not heretofore been recorded, but the importance of Dr. Donaldson's paper, and the significance of Dr. Beust's comment, led the Secretary to request Dr. Beust to prepare this abstract of his remarks.—[Ed.]

marked in the incisor region, but noticeable in the molars as well. As the incisors of the pig's mandible are not completely encased in enamel, and moreover presented some difficulties in taking accurate measurements, the transverse diameter of the posterior moiety of the first molar of sixty jaws was measured, with a Boley gauge, at the point of greatest width, which is near the amelo-cemental junction. The measurements substantiated the impressions gained on viewing the specimens, the older animals with few exceptions exhibiting an increase in width of from one-third to three millimeters in the first molars. Although at that time I acquainted Dr. Percy R. Howe, of the Research Commission, with my findings, the fact that the observations were made on only sixty jaws from various sources, and could not be checked over a period of years on a single litter of pigs, caused me to refrain from making the matter public. Personally I am convinced that there is growth in the diameters of teeth, as pointed out by Dr. Donaldson.

22. THE NERVE FIBERS AND VON KORFF'S FIBERS OF THE DENTAL PULP

Samuel W. Chase, A.M., Ph.D., School of Medicine, Western Reserve University, Cleveland, Ohio

Comparison of the results of differential staining or impregnation by several methods (Cajal, Bielschowsky, Huber-Guild, Viallane, Bethe, Dogiel, Ranvier formic, Ranvier lemon juice, Beckwith-Freud, Studnička, Vom Rath) shows that the fibers described by Mummery and others, as nerve fibers passing through the odontoblastic layer into the dentinal tubules, are not such, but are von Korff's dentinogenic fibers. The true nerve fibers in the odontoblastic layer are arranged as described by Retzius, Huber, and Rygge. Results obtained by the use of the methods listed above have failed to demonstrate nerve fibers in the dentinal tubules or dentinal matrix, excepting certain very coarse argyrophile fibers, possibly nerve fibers, which rarely pass unbranched through the odontoblastic layer, enter dentinal tubules, bend sharply, and return to the odontoblastic layer. On a basis of these results the writer is inclined to the belief that the dentine is free from nerve endings, but desires to reserve his definite opinion on this matter until the recent claims of Tojoda for his electro-osmotic method of preparing decalcified dentine for nerve impregnation have been carefully checked. The "nerve-end cells" of Mummery are not neurocytes, but are fibroblastic cells concerned with the formation of von Korff's fibers.

23. ORAL MICROÖRGANISMS OF THE DOG[10]

Theodore B. Beust, D.D.S., M.D., School of Dentistry, University of Louisville, Louisville, Ky.

The microbial films found upon the teeth of man and dog were compared. The paper deals with certain clump or matrix-forming organisms of the dog, which exhibit polymorphic developmental stages in their life cycle. An aberrant form of spirochaete and a protozoan organism, both of which are lacking in the human film, are described. The observations, made on fifteen dogs, justify the view that the dog harbors about the same number of persisting varieties as man, but that these deviate somewhat in their morphology from the human forms.

24. ADDITIONAL EVIDENCE OF THE KERATINOUS NATURE OF THE ENAMEL MATRIX

Samuel W. Chase, A.M., Ph.D., School of Medicine, Western Reserve University, Cleveland, Ohio

Specimens of decalcified developing enamel (human fetus at term) and of plantar epidermis, when similarly fixed and stained, yield evidence in support of the contention of Gies that the organic constituent of enamel is a protein of the nature of keratin. Developing enamel shows three sharply demarcated strata, which correspond strikingly as to staining reaction with the three outer strata of the epidermis. The most recently formed layer of enamel stains always like the *stratum granulosum* of epidermis. A thick stratum of developing enamel, just beneath the youngest layer, stains like the *stratum lucidum* of epidermis. The youthful enamel, lying still deeper, stains like the *stratum corneum* of epidermis. The stains imparted to comparable strata of decalcified enamel and epidermis are strikingly alike both in color and intensity. Certain colors (e.g., a characteristic vivid scarlet produced by Mallory's connective-tissue stain in the second zone of the enamel and in the *stratum lucidum* of the epidermis) are produced by similar technical treatment only in those tissues where keratinization is found. The phylogenetic and ontogenetic relationship of epidermis, oral epithelium, and enamel organ, and the known capacity of the epidermis and the oral epithelium to form keratin, render it not improbable that the enamel organ also has this capacity.

[10] Published in full on succeeding pages: *Journal of Dental Research*, 1929, ix, p. 333.

25. THE CORRELATION OF RADIOGRAPHIC AND MICROSCOPIC FINDINGS IN RESIDUAL INFECTION

John Oppie McCall, D.D.S., F.A.A.P., F.A.C.D., Dental School, New York University, New York City

The term "residual infection," as herein used, may be defined as a persistent chronic post-operative infection of the maxillary or mandibular bone. It may or may not communicate with the oral cavity, or with the external surface, by a fistula. In the absence of a fistula, diagnosis, which must depend on other signs, presents the following difficulties: the appearance and consistency of the overlying soft tissue are frequently entirely normal; there is usually no tenderness or other subjective symptom; the history, as regards post-extraction complications, is frequently negative; the radiogram and the gross findings at operation may show marked discrepancy; and there is wide variation in the radiographic findings as well. Transillumination, which has been very disappointing in the diagnosis of other jaw infections, is equally undependable in the diagnosis of residual infection.

Investigation in this field has proceeded largely along the line of correlating the findings of the surgical operation with the evidence presented by the radiogram. Bacteriologic correlation has been utilized to a limited extent: it offers certain technical difficulties which but few operators have sought to overcome. Histo-pathologic examination of tissue recovered during operation (biopsy) has apparently not been resorted to in this field. The danger of depending on the appearance of the tissues, as presented during an operation for curettage, is the wide variation in inflammatory reactions that may be found. Inflammatory reaction is a product of two forces: the virulence of the infecting bacteria, and the defensive powers of the invaded tissue. Some bacteria excite a more active defensive reaction than others; some tissues react but slightly to the presence of rather virulent bacteria.

The active organism in a large number of chronic infections of the jaw tissues is streptococcus. Of these infections the greater number are caused by streptococcus viridans. This bacterium is noted for its causation of severe infections in the jaw without inducing pain or tenderness. These are spoken of as low-grade infections. It is logi-

cal to expect that the resultant inflammation would be so mild that it would not readily be detected by any of the gross diagnostic methods ordinarily employed. Since clinical and bacteriologic experience, with teeth that show little or no radiographic evidence of infection, has indicated plainly the metastatic significance of quantitatively slight infections, it is a matter of practical as well as theoretical importance to determine the presence of infection wherever it may occur or whatever its quantitative relation.

A preliminary investigation has been made on seven cases presenting varying degrees of abnormality as indicated by the radiogram. A presumptive diagnosis of residual infection was made in each of these cases. Microscopic findings showed inflammatory reaction of varying severity. There was no correspondence between microscopic and radiographic findings as to the severity of the inflammation. Cases showing the most profound infiltration under the microscope, exhibited little aberration in the radiogram, and vice versa.

While further studies in this field should be made, it is safe to say that the views at present held by many in the dental profession as to the importance of residual infection are in need of revision.

26. A LOGICAL SYSTEM OF HAND-INSTRUMENTATION BASED ON MEASUREMENTS AND ANALYSIS OF FACTORS INVOLVED

Finn J. Bronner, D.D.S., T.L., College of Dentistry, New York University, New York City

Based on exhaustive research on factors governing the choice of angles and size of hand-instruments, three rules have been formulated by which the required angle can be quickly and accurately determined for any cavity-wall. The instrument-formula has been made more intelligible by these rules, so that the manner and position of use can be inferred from the formula-name.

The size of the blades has been determined by numerous measurements of wall-height, width, and thickness, which appear on the author's illustrated instrument chart under each operation. It is urged to consider instruments as millimeter gauges with three scraping edges. Furthermore, by applying the laws of levers there has been developed a system that counterbalances lack of finger-power.

The chart shows the instrument-selection for each class of cavity, with definite commitment on manipulation and preparation. The width and degree of the cavo-surface bevel is represented in diagrammatical form, with instruments *in situ* for the operation.

27. FINDINGS IN A CASE OF PAROXYSMAL HEMOGLOBINURIA

W. F. Swanson, B.S., D.D.S., and L. E. Van Kirk, A.B., D.D.S., Dental School, University of Pittsburgh, Pittsburgh, Pa.

The percentage of hemoglobin and the number of erythrocytes were markedly disturbed, in three rabbits, by the injection of cultures of diplo-streptococci from a pulpless tooth extracted from a patient suffering from paroxysmal hemoglobinuria.

Rabbit number 2 was injected with 6 cc. of a 16-hour culture from the root of the tooth just gingivally to the apex. Rabbit number 7 was injected with 6 cc. of a 16-hour culture from the granuloma at the apex of the tooth. Rabbit number 8 was injected with 5 cc. of a 16-hour culture from the gutta-percha filling from the root of the tooth. The accompanying graphs show the variations in the hemoglobin and erythrocytes in each case. (*Figs. 1, 2,* and *3*).

VI. FIFTH (CONCLUDING) SESSION: AFTERNOON, MARCH 24

28. A REPORT OF PROGRESS ON THE CONTROL OF DENTAL CARIES BY DIETARY AND THERAPEUTIC MEASURES

Russell W. Bunting, D.D.Sc., Dental School, University of Michigan, Ann Arbor, Mich.

The data we have accumulated during the past several years have led us to conclude that dental caries is a distinctly infective disease, and that the organism responsible for it belongs to the group known as Bacillus acidophilus. With this in mind, we have attempted to find ways and means of preventing dental caries through control of this infection. Three particular methods of accomplishing this have suggested themselves; namely, immunology, diet, and therapeutics.

(1) We have found very definite evidence that certain individuals possess an inherent immunity against the growth of B. acidophilus in the mouth. In a study of a group of such individuals we were unable to implant that organism in their mouths even after repeated

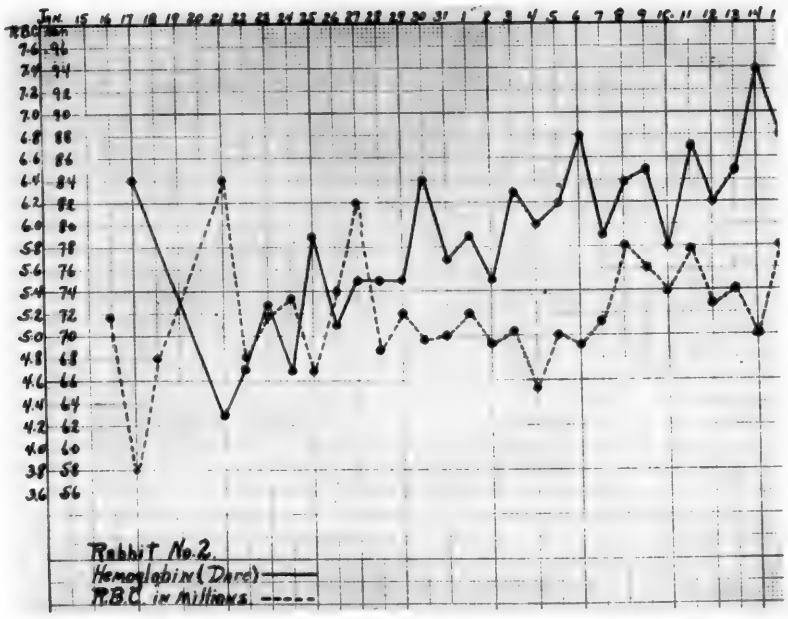

FIG. 1. Rabbit 2.—Injection: 6 cc. of a 16-hour culture from the root, gingivally to the apex.

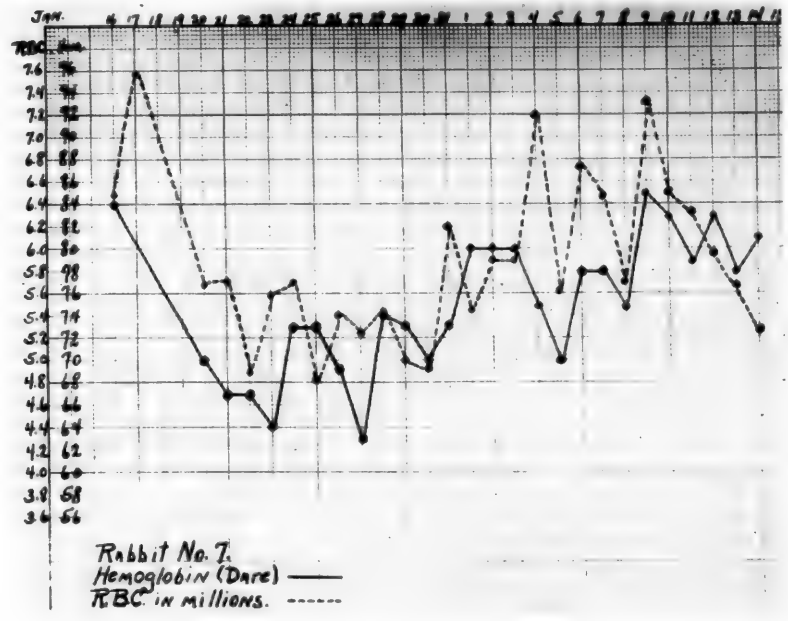

FIG. 2. Rabbit 7.—Injection: 6 cc. of a 16-hour culture from the granuloma at the apex of the root.

trials. It is possible that, in the future, ways may be found to produce an artificial immunity by which any individual can be made like those who are inherently free from this type of oral infection. At present we are unable to contribute any material progress in this direction.

(2) In both animal and human experimentation it has been found that the growth of B. acidophilus in the mouth, and the occurrence

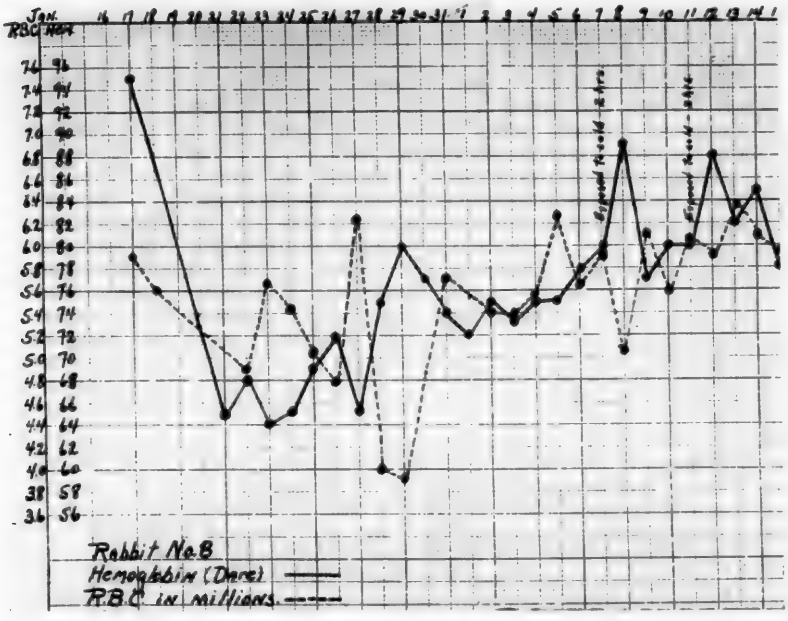

FIG. 3. Rabbit 8.—Injection: 5 cc. of a 16-hour culture from the gutta-percha filling from the root.

of caries, may be influenced by the adoption of well-balanced diets in which there is an adequate supply of the so-called protective food and a minimum of sugar. To this end we are carrying upwards of 500 children on such a diet, to note the progress of caries under these conditions. We are also aware of the fact that three groups, who are studying this question, have reported that the feeding of calcium with activated ergosterol has the effect of arresting dental caries. We are

now contemplating a study of a group of individuals thus fed, to determine the relationship of calcium feeding to the calcium in the saliva, the degree of acidophilus infection, and the activity of dental caries. We also contemplate a similar study of the effects that may be obtained by the use of activated cereals.

(3) In the study of the therapeutic control of dental caries, we have confined ourselves very largely to the use of the two drugs that seem to possess the greatest antagonism to B. acidophilus; namely, metaphen (Abbott), and hexylresorcinol (S.T. 37). At present we have an experiment under way in which we are observing four groups of children, numbering over 600, who are using hexylresorcinol as a mouth-wash twice a day. These children will be observed over a period of one year to determine how effective this preparation may be when employed in this manner.

We have made this statement for the purpose of setting before the members of the Association the line of attack we are now following, with the hope that we may receive suggestions as to the most feasible manner of proceeding further. We would welcome collaboration with any groups or individuals who are interested in, and who are working on, this problem, and will be glad to give any detailed information that may be desired.

29. PRODUCTION OF ANAPHYLACTIC RESPONSE IN GUINEA PIGS SENSITIZED THROUGH THE TEETH[11]

David Weisberger, Junior student, Harvard University Dental School, Boston, Mass.

Horse serum was introduced into cavities of the anterior teeth of female guinea pigs. In one group of the animals, the cavities were made deep enough to expose the pulp. In another group, the cavities were kept within the dentine. Normal animals, as well as animals inoculated subcutaneously with horse serum were used as controls. The animals were tested for hypersensitivity after a period of twelve days. By means of the Dale isolated-uterus method, positive anaphylactic responses were elicited in the uteri of the animals sensitized through the pulp, the dentine, and subcutaneously (control).

[11] To be published in an early issue of the *Journal of Dental Research*.

Conclusions. The teeth in guinea pigs can transmit an antigen (horse serum) to the general circulation, with consequent antibody formation, resulting in hypersensitivity in remote tissues of the body. It seems reasonable to search for possible analogies in the human body.

30. A METHOD OF SECTIONING AND DIFFERENTIALLY STAINING THE WHOLE HUMAN BRAIN[12]

P. A. Stegall, M.D., Atlanta-Southern Dental College, Atlanta, Ga.

In the treatment of this subject, the author gave a comprehensive outline of all the steps involved in this kind of work. The technic begins with the dehydration of the brain, and progresses successively through infiltration with Parowax, slicing, mounting, and finally staining. The author also described the machine used for slicing, and pointed out certain adjustments to be made on the instrument, as well as various precautions to be observed in its manipulation. The formulae for the differential stains were given, and in conclusion the pedagogic value of the method was emphasized.

31. AN OSCILLATING-ATTACHMENT FIXED BRIDGE THAT COMPLIES WITH THE REQUIREMENTS OF PHYSIOLOGICAL ORAL RESTORATION[13]

W. G. Bridge, D.M.D., Tufts College Dental School, Boston, Mass.

This attachment differs from any similar device inasmuch as it is freely movable within its limits, and without friction. All types of attachments now used on removable bridgework are retained by friction. This attachment is free to move, and immediately releases the pressure so that the tooth or soft tissue beneath is not shocked or stagnated by constant force or pressure. The means of adjustment are simple and positive, and may be varied to suit individual cases. The span or bridge proper may be constructed in various ways, using any of the stock teeth now on the market. This attachment is especially applicable to porcelain restorations, and with highly glazed

[12] A complete statement will be published in the *Proceedings of the Sixth Annual Meeting of the American Association of Dental Schools* (Chicago, March 25–27, 1929). At this meeting, the author's work was presented in detail.

[13] Published in full in the *Journal of Dental Research*, 1929, ix, p. 251 (April).

saddle and gum-block carving, sanitary and esthetic restorations can be made where surgical or accidental loss of alveolar tissue has occurred.

Let us assume, for convenience, that a loss of two bicuspids and a molar has occurred on one side of the maxilla only, with all the remaining teeth in approximate normal condition, relation, and occlusion. To stimulate circulation, and maintain a healthy condition, we decide to replace these teeth with an oscillating-attachment type of bridge. Inlay cavities are cut in the cuspid and molar for anchorage. The inlays are cast and fitted to the abutment teeth, and removed in a plaster impression. The articulation is secured and a fusible-metal model poured into the impression. The pontics are selected and ground to fit the model for esthetic relations. Plaster is built over the buccal surface to form a key to maintain this relation throughout future work.

Now we are ready to locate the position for the attachment, the latter being a heavy tube with a triangular slot through one side, in which a triangular rod slides. This is an easy fit, sliding without friction. Through the middle, at right angles, passes a lock pin, a modified screw or bolt. It is threaded at the head only, thus holding it in place without wear on the threads. The head has a square recess instead of a slot to facilitate insertion and removal. The complete attachments are placed in the desired position, and such grinding as may be necessary is done to inlay or pontic until they set vertical to the direction of greatest stress. The attachments themselves are extra heavy and will admit of considerable alteration, so it is always possible to get them into satisfactory position. When this has been done, one is waxed to the inlay with sticky wax, the screw is removed, and the outer part slipped off. This half of the attachment is then soldered to the inlay. It is now returned to the metal model, and the second one waxed parallel to it. When these are both soldered, the outer parts are placed in position, and the pontics backed, and the case completed as per type of teeth used.

When the case is polished and ready to set in the mouth, the inlays may be cemented separately and properly finished. Then the bridge proper can be adjusted to the conditions of the individual case. Vertical movement can be increased by widening the screw slot in the triangular rod to allow stimulating, functional, intermittent pressure on

the soft tissue and alveolus. The mesiodistal movement of either cuspid or molar may be varied to suit the judgment of the operator. When these adjustments are completed, the screw post is inserted, and the denture becomes a permanent fixture until its removal is desired. It can be removed at any time, without difficulty or discomfort to patient, for examination or surgical rest.

32. A CLINICO-PATHOLOGICAL STUDY OF POLYARTHRITIS AND CARDITIS SECONDARY TO ORAL FOCAL INFECTION[14]

Bissell B. Palmer, Jr., D.D.S., F.A.C.D., and Malcolm W. Carr, D.D.S., Fifth Avenue Hospital, New York City

The patient, a woman forty-seven years of age, had a series of recurrent attacks of rheumatic fever in early life, following which there was a period of remission (with but one exception) until she came to the dispensary for treatment in May 1928. The history of the present illness records three *groups* of symptoms: there were symptoms of general lowered resistance, symptoms relating to joints and fibrous tissues (polyarthritis), and symptoms indicating involvement of the cardio-vascular-renal system. The symptoms at this time were not entirely characteristic of rheumatic fever, although in many respects resembling it. The cardiac condition showed only moderate improvement with medical treatment and rest, and the polyarthritis and symptoms of general lowered resistance persisted unimproved until the patient was admitted to the hospital for complete study and observation. At this time extensive oral sepsis was revealed, and examination for foci elsewhere in the body was negative.

During this admission to the hospital the foci were eliminated and the patient given routine general care. At the follow-up examination, nine months after the patient's discharge from the hospital, there was complete relief from all the joint symptoms previously described, and a marked sustained improvement in all other subjective as well as objective symptoms. A comparative analysis of the various laboratory data indicates also that the patient had improved greatly in general health since the eradication of the oral sepsis.

We believe that the historical, clinical, and laboratory data presented in this case-report justify the conclusions on the next page:

[14] Published in full in the *Journal of Dental Research*, 1929, ix, p. 89 (April).

(1) Multiple secondary lesions may be produced by metastasis from the same primary focus, and may exist concomitantly in various tissues of the body.

(2) Symptoms of secondary lesions of focal-infectious origin probably do not manifest sustained improvement under medical treatment so long as the primary focus remains active.

(3) Oral sepsis may be the *dominant etiologic factor* in the production of lesions in the joints, muscles, and fibrous tissues, as well as the tissues of the cardio-vascular renal system.

(4) Oral sepsis may be an *aggravating* or *contributory* factor to emphasize the symptoms of lesions produced originally by rheumatic fever.

33. STERILIZATION[15]

Sterling V. Mead, D.D.S., Dental School, Georgetown University, Washington, D. C.

Keeping instruments completely immersed in boiling water for ten to fifteen minutes has been, for many years, the accepted practice in dentistry. With this method an aseptic technique has not always been possible at the dental chair. The purpose of this paper is to outline a simple and practical technique, for the sterilization of dental instruments and materials used in oral surgery, which has been adopted after careful study of all methods of sterilization. There is every reason to believe that this method can also be utilized for various dental operative procedures.

While the idea of autoclaving instruments is not new, it is the application of this principle through metal containers that has solved our individual problem. The instruments, after being washed, are placed between towels into metal containers. Everything to be used in the operation, including syringe, solution, towels for operation and patient, etc., are placed into the container. The lid is clamped down, and the instruments are autoclaved for ten minutes at eighteen to twenty pounds pressure. After sterilization, the instruments are placed in the operating room on a table. When ready for use a nurse takes off the lid, and the operator, after having "scrubbed up," removes the

[15] To be published in an early issue of the *Journal of Dental Research.*

instruments by taking hold of the bottom towel and placing the group on the table. In this way the instruments are brought from the sterilizer to the patient without contamination, and no one ever touches the instruments excepting the operator, unless the operation requires the assistance of a sterile assistant.

34. CONCERNING THE CIRCULATORY SYSTEM IN THE DENTINE

Charles F. Bödecker, D.D.S., F.A.C.D., School of Dental and Oral Surgery, Columbia University, New York City

Dentine is composed of a calcified basis-substance, and innumerable tubules containing the dentinal fibrils or soft fibers of Tomes. The author has shown that the dentinal fibril is a *tube* and not a "fiber,"[16] by first emptying it of its protein-bearing lymph before fixing. This fact is important in appreciably increasing the permeability of the dentine, for tubes can transport a greater amount of lymph than "fibers." The author has resumed his investigations in this field because he believes he has discovered a new factor in dental caries that constitutes the predisposing cause of this disease. He believes that qualitative and quantitative variations in the "dental lymph" are responsible for the susceptibility and immunity of the teeth to decay.

35. A SIMPLIFIED PROCEDURE FOR THE SEPARATION AND IDENTIFICATION OF PROTEIN IN DENTAL ENAMEL

Theodor Rosebury, D.D.S., and William J. Gies, Ph.D., F.A.C.D., Laboratory of Biological Chemistry, Columbia University, New York City*

Preparation of enamel. Freshly extracted teeth, thoroughly cleaned with a stiff brush and soap under cold water, are immersed for about thirty seconds in 5 per cent hydrochloric acid solution, and again scrubbed. A series of longitudinal grooves is cut on the axial surface of each crown, under running cold water, with a separating disc in an

[16] Bödecker: "Some recent investigations upon the histology of the dentine;" *Dental Review*, 1912, xxvi, p. 260. "The soft fiber of Tomes a tubular structure;" *Dental Cosmos*, 1914, lvi, p. 1378. "The soft fiber of Tomes a tubular structure," etc.; *Journal of the National Dental Association*, 1922, ix, p. 281.

* Dr. Rosebury is the William J. Gies Fellow in Biological Chemistry at Columbia University, in 1928–29.

engine. The grooves are cut to a depth of about 0.5 mm. at the thickest portion of the enamel. Carious areas and abraded surfaces, and all suspicious surface deposits or discolorations that cannot be removed with a stone under cold water, are avoided. The grooved tooth is again scrubbed, then dried with filter paper. Enamel is removed from the edges of the grooves with a straight chisel. To avoid contamination of the product with scurf from the head or hands, a cap and rubber gloves are worn during the chiseling. By confining the grooves to intact areas of enamel, and terminating them well within the cervical limit of the enamel, freedom of the product from dentine and cementum is assured. (This is verified by microscopic examination.) Fragments of Nasmyth's membrane and other surface contaminants, are excluded by the thorough preliminary cleansing. At least four teeth are used for the preparation of a single specimen of enamel.

Demineralization. The enamel powder thus prepared is demineralized in an apparatus consisting of a tall glass cylinder, of about 400 cc. capacity, closed with a rubber stopper perforated to hold vertically a short length of half-inch glass tubing. A transparent collodion bag, made on the inner surface of an 18 mm. test tube and cut short, is tied, with cotton thread, to the lower end of the glass tube, which protrudes a short distance above and below the stopper. The pulverulent enamel is placed within the collodion bag, and the bag and cylinder are filled with acid solution to the same level. Dilute acetic acid is useful for the purpose, having comparatively little tendency to denature the protein. The assembly is covered with an inverted beaker as a dust cap. The acid in the *cylinder* is changed at intervals by lifting the stopper and bag, and decanting. The exterior acid diffuses into the collodion bag, the dissolved mineral salts diffuse outward, and the enamel residue remains within, where portions may be readily obtained with a pipette as needed. The progress of demineralization may be followed by testing the fluid in the cylinder at intervals for phosphate. A concentration of 1.5 per cent acetic acid, renewed occasionally, with the bag at the upper level of a high column of solution, is effective in about ten days.

Tests applied to the organic residue (*"matrix"*). The following tests were employed to determine the identity of the demineralized residue, in each case comparative tests having been made on a variety of sub-

stances of known chemical nature: (1) An examination of the physical characteristics of the residue: gross and microscopic. (2) The biuret test, under the microscope, using Gies' biuret reagent. (3) The Millon test, in a test tube, on a microscope slide, or in a pipette used to remove particles from the collodion bag. (4) The xanthoproteic test, on a microscope slide. (5) The effects of acid and alkaline reagents, observed microscopically with the aid of an eyepiece micrometer. (6) Digestive action of pepsin, followed by (7) digestive action of trypsin. (8) The lead test for unoxidized sulphur, in a test tube, on a large quantity of residue. (9) Differential staining reactions with modifications of the Mallory and Pianese stains.

Results. The general results already obtained are these: (1) The presence of protein in human enamel has been confirmed.[17] (2) The organic residue contains protein that belongs to the albuminoid group. (3) It shows the distinguishing characteristics of keratin, and (4) seems to be closely allied to neurokeratin. (5) The liquid within the collodion bag would contain any associated *soluble though indiffusible* protein. Biuret tests heretofore applied to this liquid have been negative. (6) Similar tests for *soluble though diffusible* protein, in the dialysate, have also been negative. (7) The enamel keratin, treated with sodium sulfid solution, turns green, but whether this is due to impurity, such as iron, has not yet been determined, although numerous sources of such error have been excluded.

36. FURTHER EVIDENCE THAT THERE IS NO METABOLISM IN ENAMEL OF FULLY FORMED TEETH

William J. Gies, Ph.D., F.A.C.D., Laboratory of Biological Chemistry, Columbia University, New York City

Various destructive changes that occur in the enamel of *fully formed* human teeth have been ascribed to abnormal nutrition. A tendency erroneously to regard direct chemical change by simple diffusion as "metabolism," or evidence of "nutrition," has been noted.[18] The proof that enamel contains albuminous matter[17] has induced

[17] Bödecker and Gies: *Proceedings of the Society for Experimental Biology and Medicine*, 1924, xxii, p. 175. Also Gies: *Journal of Dental Research*, 1926, vi, p. 143.

[18] Gies: *Journal of Dental Research*, 1926, vi, p. 146.

some investigators to assume that its presence indicates nutritional capacity in the enamel, regardless of the nature of the protein and despite the fact also that, although permeable to water and various other substances, enamel completely lacks power of self-repair after injury and has no cellular nuclei. That the contained protein, chiefly if not wholly keratin, is not associated with *intrinsic* nutritional changes in the enamel of *fully erupted* teeth is indicated by the present writer's further observation that conventional tests for digestive and tissue enzymes, in pulverized normal enamel, have been completely negative.

VII. Executive Data

A list of the officers, and new members, elected March 23, 1929[19]

a. Officers (1929–30)

President—Arthur D. Black, Northwestern University. *President-elect*—U. G. Rickert, University of Michigan. *Vice-president*—A. E. Webster, University of Toronto. *Treasurer*—William Rice, Tufts College. *Secretary*—William J. Gies, Columbia University.

b. New members

Boston, Mass.

Benjamin Tishler, 358 Commonwealth Ave.

Chicago, Illinois

Howard C. Benedict, 311 E. Chicago Ave.
Earl P. Boulger, 17 S. Crawford St.
Edgar D. Coolidge, 25 E. Washington St.
Ralph H. Fouser, 58 E. Washington St.
Stanley D. Tylman, 185 N. Wabash Ave.

Halifax, N. S.

Raymond J. Bean, 103 College St.
E. Gordon Young, Dalhousie University.

[19] A list of the officers and members as of November 15, 1928, was published in the *Journal of Dental Research*, 1928, viii, pp. 697–699. No new members were elected after November 15, 1928 and before March 23, 1929.

Minneapolis, Minnesota

Joseph T. Cohen, 431 Syndicate Bldg.

J. F. McClendon, 311 Millard Hall, University of Minnesota.

New York, N. Y.

I. Hirschfeld, 730 Fifth Ave.

Philadelphia, Pa.

W. G. Downs, Jr., Medical School, University of Pennsylvania.

Samuel E. Pond, Medical School, University of Pennsylvania.

Vienna, Austria

Bernhard Gottlieb, Türken Str. 15.

Ernst Kellner, Türken Str. 15.

Emerich Kotanyi, Kirchengasse 17.

Rudolf Kronfeld, Bognergasse 7.

Moriz Leist, Jörger Str.

Arthur M. Schwarz, Türken Str. 15.

Georg Stein, Morzin Pl. 5.

Josef Weinmann, Frankgasse 1.

Herman Wolf, Schönborngasse 9.

B. NAMES OF MEMBERS PRESENT AT ONE OR MORE SESSIONS

(Arranged in sectional groups)

Ann Arbor.—Bunting, Rickert, Ward. *Boston.*—Miner, Morse (A. L.). *Chicago.*—Arnold, Benedict, Bergeim, Black, Blayney, Boulger, Coolidge, Davis, Fouser, Hatton, Mueller, Nichols, Noyes, Nylander, Orban, Petersen, Potts, Sippy, Skillen, Tylman, Welker. *Halifax.*—Thomson. *Minnesota.*—Best, Gardner. *New York.*—Dunning (W. B.), Gies, Hyatt, Leonard, McCall, Owre, Palmer, Waugh. *Philadelphia.*—Appleton. *Pittsburgh.*—Brand, Dewey, Friesell (H.E.), Van Kirk. *Toronto.*—Webster. *Not yet members of a section.*—Beust, Bryan, Chase, Jay. Total—47.

At each scientific session there was a large attendance of visitors, many of whom were members of faculties of dental schools that were represented at the succeeding annual meeting of the American Association of Dental Schools.

MECHANISM OF INTOXICATION BY INTESTINAL ABSORPTION IN CHRONIC ORAL SEPTIC FOCI[1]

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I. INTRODUCTION

Dental sepsis, being a visible lesion and one that interferes with proper nutrition, must have interested man since the beginning of his existence. We read in the vulgate edition of the bible that "Moses was 120 years old when he died; his eye was not dimmed, nor were his teeth loosened" (Deuteronomy, XXXIV, 7). From this special mention of the absence of dental septic processes, one would be led to infer that these lesions were of special interest to the oriental races at an early period. Hunter (1899) laid stress upon the influence of dental sepsis on toxic and infective gastritis. Hurst (1923) found a heavy coccal flora in the duodenum in a large proportion of the dental septic cases examined. This observer, like many others that could be cited, explained this finding on the theory of a toxic gastritis produced by swallowing the septic material from the oral cavity. The explanation given by this school of thought is about as follows: The acid in the stomach has a bactericidal action upon the swallowed microbic material. This germicidal action is overwhelmed by the ingestion of large amounts of septic material. As a consequence the gastric mucosa

[1] Read at a session of the Seventh Annual Meeting of the International Association for Dental Research, Northwestern University Dental School, Chicago, Ill., March 23–24, 1929. See abstract: *Journal of Dental Research*, 1929, ix, p. 279.

secretes less acid owing to the toxicity of the pus swallowed from the oral lesion. This reduces the bactericidal action and a vicious cycle is thus set in motion. The presence of cocci and other bacteria in the duodenum is attributed to the hypochlorhydria, or even achylia, that is usually present in chronic oral-sepsis patients.

We do not agree with this explanation. Our experimental work has led us to believe that the true explanation of the gastro-intestinal dysfunction, and the systemic intoxication, is more complicated and is a part of a general biological picture of adaptation to certain abnormal environmental factors. There are three important environmental factors to which an animal organism must adapt itself if it is to survive. These three factors are meteorological (including temperature, humidity, wind velocity, sun-light, barometric pressure, etc.), nutritional, and lastly parasitic in the broadest sense. The nutritional factor has received most attention from students of odontology. The climatic or meteorological factor is of equal importance in maintaining a normal individual.

There is a certain balance or equilibrium between the skin and superficial areas of the body, and the internal or splanchnic region. Normally we have a certain number of white blood-cells per unit volume of blood in the capillaries of the skin in the post digestive resting physiologic state of metabolism. Under these conditions we have the same number of leucocytes in the blood in the internal organs. There is a delicate equilibrium between these two systems. Under the above conditions, that is six to eight hours after a meal and lying quietly in bed, we are as nearly at rest as is possible. If we stimulate the skin, with ultra-violet light, heat, mechanical friction, etc., these conditions are entirely changed. There is hyperemia of the skin, leucocytosis, increase in permeability of the skin (blister fluid analysis), and increase in function of the adjacent peripheral glands. At the same time there is relative leucopenia in the splanchnic region, diminution in the gastric secretion, contraction of the stomach, diminished flow of bile, decreased permeability of the splanchnic glandular tissue (as determined by thoracic-duct lymph analysis). In other words there is an opposing action between the splanchnic and peripheral systems. When one is stimulated, the other is depressed, and vice versa.

II. METHODS AND RESULTS

The upper half of the small intestine in the normal post-digestive animal is slightly acid in reaction. In this zone we find few bacteria, which are simple diplococci belonging to the enterococci group. The lower third of the small intestine is neutral or alkaline in reaction. In this zone we find a rich and varied bacterial flora (*Table 1*). *Table 2* indicates the changes that take place in fever. The same results

TABLE 1

Bacterial flora and H-ion concentration of the small intestine (post-digestive) of healthy dogs

PORTION OF SMALL INTESTINE	BACTERIAL FLORA	H-ION CONCENTRATION
		pH
Duodenum	Few Gram-positive cocci	5.0 to 6.0
Jejunum, upper half	Few Gram-positive cocci	5.5 to 6.5
Jejunum, lower half	Gram-positive cocci; few Gram-positive and Gram-negative bacteria	6.0 to 7.0
Ileum	Very rich bacterial flora	6.8 to 8.0

TABLE 2

Bacterial flora and H-ion concentration of the small intestine of fever dogs

PORTION OF INTESTINE	BACTERIAL FLORA	H-ION CONCENTRATION
		pH
Duodenum	Heavy growth, mixed flora	6.9
Upper jejunum	Heavy growth, mixed flora	7.2
Lower jejunum	Heavy growth, mixed flora	7.4
Ileum	Heavy growth, mixed flora	7.5

are brought about by any condition that causes a diminution in gastric secretion. *Table 3* shows the changes that occur in the duodenal flora, and the distribution of leucocytes, when this segment of the alimentary tract is made alkaline in reaction. Any condition that leads to an alkaline reaction in the upper part of the small intestine causes the appearance within the lumen of this organ of bacterial flora of large-intestinal type. This may seem a small and unimportant matter. Important biochemical reactions take place within the upper ten to

fifteen feet of the small intestine. In all of the animals so far examined there is an acid reaction in the upper level of the intestinal tract and a scanty bacterial flora—this includes the guinea-pig, rabbit, rat, mouse, pig, horse, cow, sheep, dog, and man.

The upper part of the intestinal tract loses its power to disinfect itself if the contents becomes alkaline. *Fig. 1* shows the results obtained in dogs. The duodenum was fixed to the anterior abdominal

TABLE 3

Changes in the bacterial flora of the duodenum and the peripheral leucocytes after alkalinization of the duodenum[1]

BACTERIAL FLORA	TIME	TEMPERATURE	WHITE BLOOD CELLS	DIFFERENTIAL COUNT					ARNETH'S INDEX
	a.m.								
Dog VI[2]	9:50	101.2	9,200						
Before injection	10:30	101.2	18,600	85	12	1	1	1	36.0
Four colonies enterococci	11:00	101.4	18,800	87	9	2	2		26.0
After experiment	11:30	101.0	19,000	94	3	1	2		34.5
150–200 colonies, mixed flora	12:00	100.8	21,200	87	12		1		52.5
Dog VII[3]	8:20	103.4	35,500	90	7		1		45
Before injection	9:00	102.4	29,600	87	9	1	1		42
150–200 colonies, mixed flora	9:30	102.8	43,200	91	8				48
After experiment	10:00	102.6	36,200	88	9	1	2		53.5
200–250 colonies, mixed flora	10:30	102.8	41,300	92	5	3	1		33.5

[1] Fifty cc. of a phosphate buffered solution (8.9 pH; 37°C.) injected into duodenum.
[2] Dog VI injected at 10 a.m.
[3] Dog VII injected at 8:30 a.m.

wall and a fistula established through the appendix [Arnold-1926, Arnold and Brody-1926 (1), (2)] under the usual sterile surgical procedures. After the wounds healed, suspensions of B. prodigiosus, B. pyocyaneus, B. typhosus, B. paratyphosus, in normal salt-solution, were injected directly into the duodenum. The concentrations were recorded at definite time intervals in the caecum. In other experiments the bacteria were suspended in alkaline buffered phosphate solution (8.0 pH) and injected as indicated above. The curves show that the alkaline solution interferes to a great extent with the auto-

sterilizing action of the small intestine. *Fig. 2* shows the results of some experiments carried out on similar preparations, except that these animals had non-leaking gastric fistulae in addition to the an-

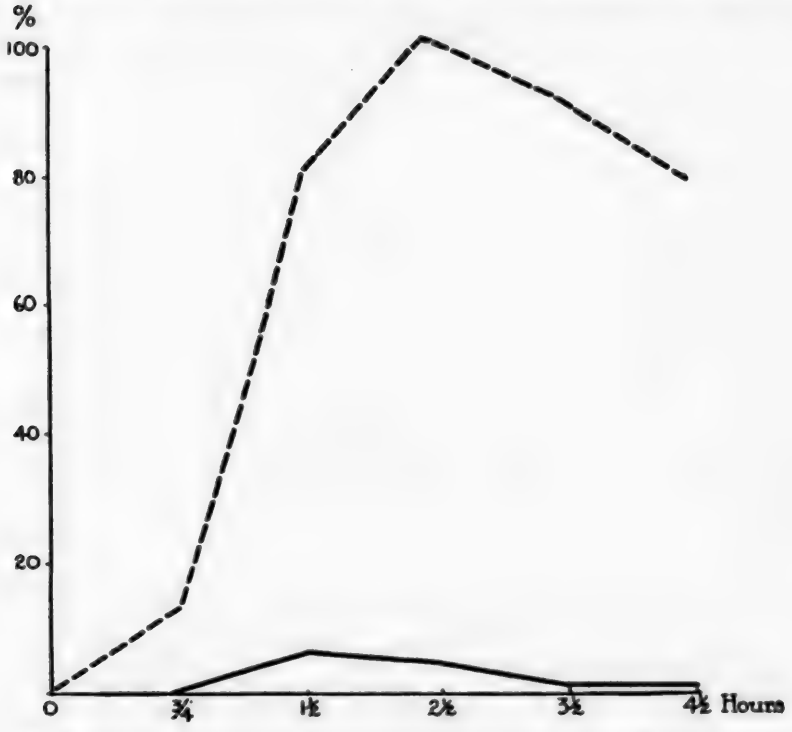

FIG. 1. GRAPH SHOWING THAT ALKALINITY INTERFERES TO A GREAT EXTENT WITH THE AUTO-STERILIZING ACTION OF THE SMALL INTESTINE

The duodenum was fixed to the anterior abdominal wall and a fistula established through the appendix. See also *fig 2.*

Ordinate.—Percentage of viable bacteria appearing in the caecum after intraduodenal injection. *Abscissa.*—Time caecal specimens were taken after injection into duodenum. *Continuous line.*—One agar slant of B. prodigiosus suspended in 20 cc. of saline. *Broken line.*—Same bacteria suspended in alkaline buffer phosphate solution (8.5 pH).

teriorly fixed duodenum and caecal fistulae. Plain milk plus the bacteria were fed by mouth. Gastric acidity, hydrogen-ion concentration in the duodenum, and proportion of ingested bacteria appearing

in the caecum are indicated. *Fig. 3* shows results of a similar experiment, except that the milk was made alkaline (8.0 pH) to increase the gastric load. These graphs (*figs. 1–3*) illustrate the loss of bactericidal power during a period of alkalization of the upper level of the small intestine (Arnold and Hull-1928).

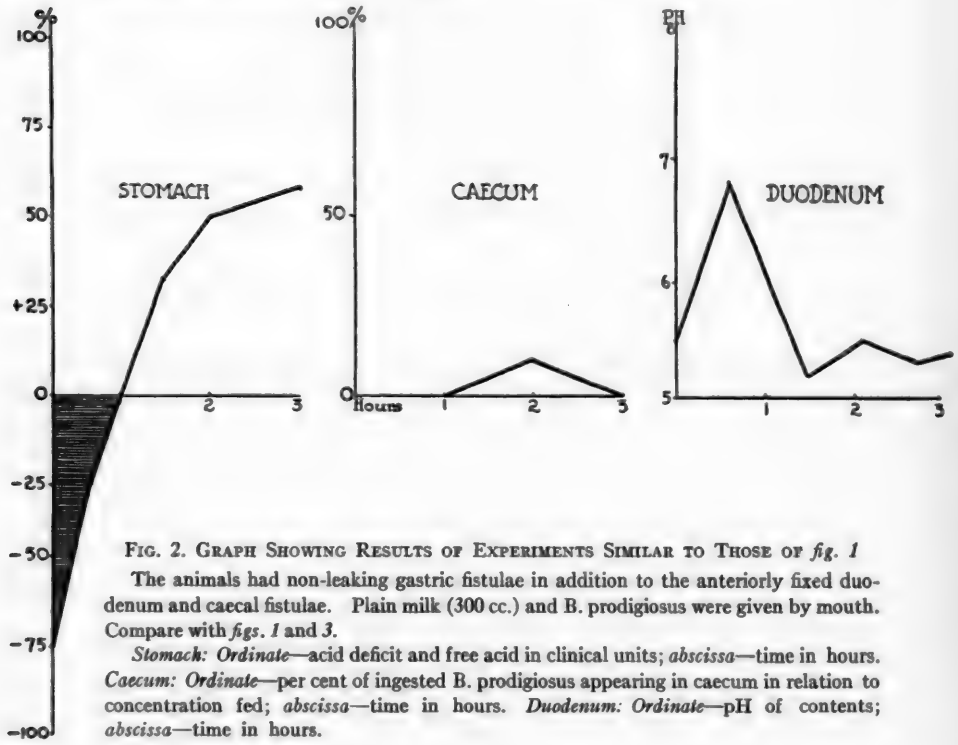

FIG. 2. GRAPH SHOWING RESULTS OF EXPERIMENTS SIMILAR TO THOSE OF *fig. 1*

The animals had non-leaking gastric fistulae in addition to the anteriorly fixed duodenum and caecal fistulae. Plain milk (300 cc.) and B. prodigiosus were given by mouth. Compare with *figs. 1* and *3*.

Stomach: Ordinate—acid deficit and free acid in clinical units; *abscissa*—time in hours. *Caecum: Ordinate*—per cent of ingested B. prodigiosus appearing in caecum in relation to concentration fed; *abscissa*—time in hours. *Duodenum: Ordinate*—pH of contents; *abscissa*—time in hours.

We have shown that sudden changes in reaction of the contents of the duodenum and jejunum can be closely associated with permeability of the gut-wall for viable bacteria. Under a local anesthetic, the thoracic duct in the dog was cannulated and the duodenum exposed. The lymph was collected in five-minute samples in sterile tubes. Living bacteria were injected into the lumen of the duodenum, and the lymph from the thoracic duct cultured to determine their presence.

When distilled water, normal salt solution, or acid-buffered phosphate solutions, were used to suspend the bacteria, the lymph remained sterile. When alkaline phosphate solutions were used, bacteria injected into the lumen of the duodenum could be cultured in the thoracic-duct lymph. *Fig. 4* shows the results of such an experiment.

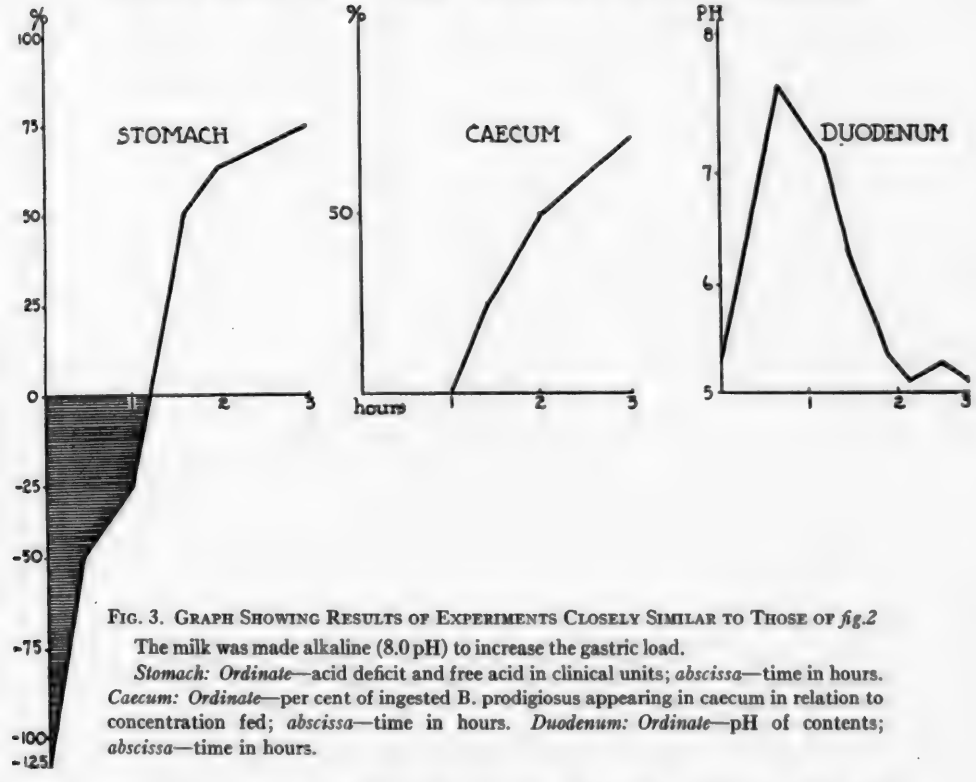

FIG. 3. GRAPH SHOWING RESULTS OF EXPERIMENTS CLOSELY SIMILAR TO THOSE OF *fig.2*

The milk was made alkaline (8.0 pH) to increase the gastric load.

Stomach: Ordinate—acid deficit and free acid in clinical units; *abscissa*—time in hours. *Caecum: Ordinate*—per cent of ingested B. prodigiosus appearing in caecum in relation to concentration fed; *abscissa*—time in hours. *Duodenum: Ordinate*—pH of contents; *abscissa*—time in hours.

The concentration of leucocytes per cmm. of blood collected from the ear of the animal is indicated by the continuous line on this chart. It will be seen that the permeability of the splanchnic system, for bacteria placed in the lumen of the duodenum in a healthy animal, is associated with sudden changes in the distribution of the leucocytes in the circulation. This is not the place to discuss changes in the autonomic nervous equilibrium in the body brought about by intra-

intestinal stimulation. At this time we only call attention to this reaction as an example of changes in intestinal permeability to viable bacteria. The whole question has been recently reviewed in detail by the author (Arnold–1929).

In experimental avitaminosis, there is a change in the response of the glandular elements of the gastro-intestinal tract to food and other

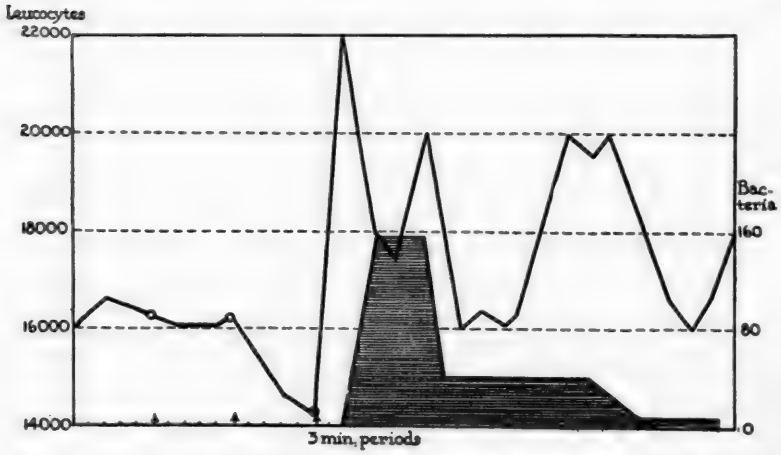

FIG. 4. GRAPH INDICATING THAT THE PERMEABILITY OF THE SPLANCHNIC SYSTEM FOR BACTERIA, PLACED IN THE LUMEN OF THE DUODENUM IN A HEALTHY DOG, IS ASSOCIATED WITH SUDDEN CHANGES IN THE DISTRIBUTION OF THE LEUCOCYTES IN THE CIRCULATION

Ordinate.—Left: number of leucocytes per cubic millimeter of blood from dog's ear. *Right:* number of viable bacteria appearing in thoracic-duct lymph after intraduodenal injection. *Abscissa.*—Time in three-minute intervals. *Continuous line.*—Leucocytes. *Arrows—First:* time peritoneum was opened; *second:* time one agar slant of B. prodigiosus in 20 cc. of saline was injected into lumen of duodenum; *third:* time same bacteria suspended in 20 cc. of alkaline buffer phosphate solution were injected intraduodenally. *Heavy-lined area.*—Number of viable bacteria appearing in each cubic centimeter of thoracic-duct lymph.

stimuli. Associated with the diminished functional response of the gastric mucosa is a hypochlorhydria. There is a loss of power of the upper part of the small intestine to regulate bacteria within its lumen. It is reasonable to expect that one of the important predisposing factors to body invasion is the loss of the power of a body surface to destroy microbic life placed in contact with this covering layer. This

problem is a fundamental one and at this time can only be touched upon in passing.

Calcium and other inorganic salts that play an important rôle in odontological problems are insoluble in neutral or alkaline media. The whole inorganic-salt metabolism can then be altered by changes in the reaction of the contents of the absorbing segments of the alimentary tract. Beyond the normal variations within physiological limits of gastric secretory function, certain rather clearly defined pathological functional changes are well known. A diminution in the power of the gastric mucosa to concentrate acids within its lumen (hypochlorhydria) as well as an apparent loss of this function (achylia) are common clinical conditions. Van der Ries (1925) has reviewed the literature upon the bacterial flora and H-ion concentration of the gastro-intestinal tract under normal and certain pathological conditions. In hypochlorhydria, there is a gradual ascension, cephalad, of the flora of the large intestine, the extent depending in part at least upon the relative deficiency of acid. This of course is influenced by the diet. Preponderance of basic substances in the diet leads to a greater proportional deficiency of acid radicals than an acidified diet. Sour-milk therapy of all kinds must be reviewed from this standpoint. Bacteriologically there is some question as to whether it is possible, by ingesting B. acidophilus, to cause an implantation of these bacteria in the large intestinal flora. Acidophilus-milk therapy is combined with lactose therapy plus an acidified food. According to Britten and Thompson (1925), retrogressive lesions within the oral cavity increase in relative incidence after the fortieth year. These observers have found that cardio-vascular and other degenerative diseases show the same tendency. There seems to be a progressive decrease in hydrochloric acid secretion by the stomach after the fortieth year of life (Friedenwald and Morrison-1926; Keefer and Bloomfield-1926). The gradual increase in acid-deficit in the gastric content causes a change in the endogenous bacterial flora of the small intestine, and interferes with inorganic-salt metabolism. In achylia this condition is more marked, the fecal flora extending even into the lumen of the stomach, and the contents of the whole gastro-intestinal tract is alkaline in reaction.

Evans, Riding and Glynn (1927), as well as others, have shown that

there is a lowered sugar tolerance in many patients with oral sepsis. These patients are not true diabetics, inasmuch as the carbohydrate tolerance returns to normal when the foci are removed and healed. This is an instance of systemic intoxication due to a chronic focus of infection. Change in the oral cavity during the course of pregnancy has been a common observation. Kramer-Petersen (1919) and Nakai (1925) have shown that there is a diminution in the amount of acid secreted by the stomach in many pregnant women, especially when there is evidence of toxemia. Andresen (1922) has shown a lessened response of the stomach in chronic diseases of the pelvic organs in the female. Septic oral lesions have been held by some observers as a definite etiological factor in pernicious anemia. There is a condition of achylia in this disease. Septic oral foci are encountered in most cases of pellagra. Guthrie (1926) has shown that the absence of free acid in the gastric contents is an important part in the diagnosis of pellagra.

Faber (1926) has given us the best workable classification of the pathogenesis of achylia gastrica and allied gastric hypofunctional conditions. Faber considers that the mucosa and its secretory apparatus respond to intoxications like any other parenchymatous organ, such as the kidney, pancreas, liver, etc. Recuperation after removal of the toxic foci depends upon the extent of the damage done. Further discussion of this question would take us beyond the field of the present communication. The importance of maintaining a proper functioning gastric mucosa has already been emphasized. The acid-base balance of the contents of the absorbing intestinal tract is of great importance to the student of odontology. Inorganic-salt metabolism is inseparably connected with the reaction of this part of the intestinal tract. Biological control of the endogenous bacterial flora of the alimentary tract is also intimately interwoven into this problem.

New light has been thrown upon an old problem, if certain parts of our work are considered. The capillaries of the lung are the first vascular capillary bed that material absorbed from the intestinal tract by way of the thoracic duct encounters in the body. It must follow, then, that the lungs act for lymph-absorbed material, as does the liver for blood-absorbed material, from the alimentary tract. As mentioned above we have shown that viable bacteria can be found in

the lymph stream just before it enters the venous system. Lung infections and mediastinal lymphatic hyperplasia, which so often accompany or are sequelae of oral sepsis, can well be of intestinal origin rather than due to a travelling of the infection down the respiratory tract to the lung. This is only one of the many problems that are reopened, from a new angle, by considering oral sepsis from the standpoint of this paper.

When B. coli are incubated in broth for forty-eight hours and passed through a Berkefeld filter, the filtrate contains a heat-labile, neurotropic, toxic substance. This sterile filtrate, injected intravenously into rabbits (4 cc. per kilo), causes convulsions and death within a few hours. When B. coli are incubated in broth for fourteen days, and passed through a Berkefeld filter, the filtrate contains a relatively heat-stable, enterotropic, toxic substance. This sterile filtrate, injected intravenously in rabbits (4 cc. per kilo), was not followed by any immediately noticeable results. After nine to eighteen hours all of these animals developed a diarrhea and died several days later. Twelve animals have been used with each of these two types of toxic filtrates. The exotoxin, or neurotropic substance, is more variable in its reaction. The endotoxin, or enterotropic substance, has given uniform results in our experiments. The *exotoxins* were given by mouth (2.5 cc. per kilo), every three days for thirty days. The rabbits did not show any change during the two months of observation. When the same amount of *endotoxin* was administered by mouth, the results were entirely different. After four weeks there was diarrhea, and in eight to ten weeks the animal died. The total loss of weight was about 25 per cent. This is significant, in that we have used young animals that should increase in weight upon the carrot and cabbage diet they were eating. *Fig. 5* shows the results of one experiment in which three such animals were given B. coli endotoxins by mouth. Plantenga (1925), in Holland, carried out almost the same experiment upon young calves and obtained similar results. In some of our experiments we gave intravenously or by mouth a minimal dose of the enterotropic toxic material to cause a colitis, and the animals were allowed to recover. Then, after a few weeks, another dose was administered, either intravenously or per os, and in each instance a colitis was produced. We feel safe

in saying that no immunity is produced. The severity and duration of the chronic colitis can be controlled to a great extent by dosage per

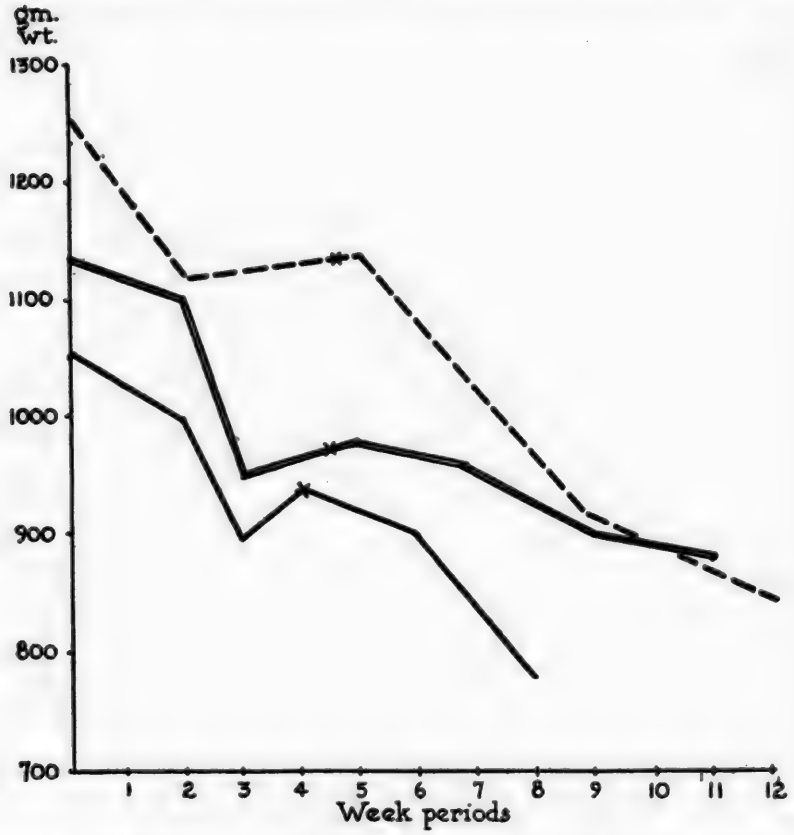

FIG. 5. GRAPH SHOWING EFFECTS OF B. COLI ENDOTOXINS GIVEN, TO THREE DIFFERENT RABBITS, PER OS EVERY THREE DAYS FOR FIVE WEEKS

Each line represents the weekly weight of one rabbit. "X" on each line indicates the time of appearance of diarrhea.

Ordinate.—Weight of rabbits in grams. *Abscissa.*—Time in weeks after beginning of experiment.

os of the sterile enterotropic toxic coli substance. Even intermittent colitis can be experimentally produced under proper conditions.

It may be seen, from the above experiment, that B. coli toxins were absorbed through the intestinal tract when fed by mouth, and produced the same symptoms and pathology in the experimental animal as those caused when these toxins were injected intravenously. The B. coli used in these experiments were isolated from rabbit feces, and cannot be considered foreign strains. Loss of weight, followed by chronic colitis, resulted after repeated oral administrations of B. coli toxins. No diarrhea occurred in our rabbit colony during the course of these experiments. In view of what has already been presented in this paper, concerning the changes in the enteric bacterial flora in chronic intoxications, the persistence of B. coli in the absorbing end of the small intestine is important. It may not be inferred that chronic colitis should follow in every case of ascension of the B. coli flora into the jejunum and duodenum. All abnormal physiological functions are attempts on the part of the host to adapt itself to the existing conditions. The intoxication may be manifested in a variety of different ways.

III. DISCUSSION

The upper half of the alimentary tract can serve as a port of entry for toxic substances. Under normal physiological conditions this part of the intestinal canal is a highly specialized digesting and absorbing area. Under abnormal physiological conditions, such as those induced by certain chronic septic foci, and the accompanying systemic intoxication, this portion of the digestive tube can be so changed that chemical and bacterial toxic material passes through it into the body. It should be borne in mind that the acidified milk therapy, such as buttermilk, bulgaricus milk, or acidophilus milk, used to control putrefaction in the large intestine, is the ideal form to correct upper-intestinal toxin absorption. When such acidified foods are ingested, they tend to compensate for the lack of gastric-acid secretion and thereby aid in restoring the normal acid-base balance in the lumen of the upper part of the intestinal tract.

The return to normal, after removal of chronic septic foci in the oral cavity, is dependent upon the amount of damage done to the glandular tissue in the gastro-intestinal tract as well as the age of the patient. The acid secretion of the stomach tends to diminish pro-

gressively after the fortieth year of life. When the gastric acidifying mechanism is below par, then the food must be changed or there will be an alteration in the endogenous bacterial flora, the H-ion concentration, acid-base balance, and other reactions within the lumen of the upper half of the small intestine. These conditions might be regarded as unimportant inasmuch as the period of maximum dental demand for inorganic salts has passed. But in reality it represents an important period for the age-group of the population that seek relief from intoxications of oral origin.

We consider the changes that take place within the lumen of the intestinal tract, such as a migration cephalad of the fecal flora and an increase in the alkaline reacting substances, as a part of the biological reaction of the organism to certain abnormal stimuli.

IV. SUMMARY

Intra-intestinal chemical and bacteriological changes can take place as a result of systemic intoxication due to oral septic lesions. These changes are brought about by damage to parenchymal tissue in the gastro-intestinal tract. The diminution in acid secretion by the stomach tends to upset the normal acid-base balance of the contents of the upper half of the small intestine. This is accompanied by an ascension upward of fecal bacterial flora into the jejunum and duodenum. The alterations in inorganic salt metabolism, and the absorption of by-products of disintegrated B. coli proteins, have been emphasized.

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BIOCHEMICAL STUDIES OF STREPTOCOCCI

III. Availability of Carbon Compounds[1]

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I. INTRODUCTION

Although none of the synthetic media mentioned in this paper have supported the continued growth of streptococci, many of them may be used successfully for testing the value of various substances as nutrients. The effect of addition of any substance, whether beneficial or detrimental, may be most easily observed in a medium that just barely supports life in the organism. This is equally true of media that just fail to permit growth.

We studied the effect, on the growth and viability of streptococci, of the addition of various carbon compounds to the media. The details of procedure are those given in the second paper in this series.[1]

II. RESULTS

1. *Dextrose.* The value of dextrose as a nutrient for streptococci is indicated in *Tables 1–5*. These experiments show that dextrose markedly shortened the period of viability and hence did not support

[1] The previous papers in this series were published in the *Journal of Dental Research:* (1) Krasnow and Gies, 1929, ix, p. 29; (2) Krasnow and Rosenberg, *Ibid.*, p. 123.

[2] A preliminary report was published in the *Proceedings of the Society for Experimental Biology and Medicine*, 1928, xxv, p. 295.

growth (*Tables 1, 2, 3, 4*). The results recorded in *Table 5* show an apparent increase in viability owing to the addition of dextrose, but the increases are so slight that they may be without significance.

The data obtained in comparisons between dextrose and other carbohydrates, or related compounds, support the above findings. The relative values for viability and growth may be expressed by the series below (> = more beneficial than):

a. Sucrose > lactose / maltose > raffinose > dextrose / mannite (*Table 7*)

b. Sucrose > maltose / lactose > galactose > dextrose (*Table 6*)

c. Sucrose > galactose > dextrose / maltose / lactose (*Table 6*)

Data for growth in media containing these carbohydrates are given in *Tables 6* and *7*.

There are several references in the literature to the growth value of dextrose. Samkow (11), and Waksman and Jaffe (13), state that although dextrose was harmless it gave little aid. Frankel and Pyle (2) showed that dextrose retarded growth slightly. Koser and Rettger (7) reported that glycerol was better than dextrose, because less acid was formed and the organism was therefore "freer to develop." None of these investigators refer specifically to streptococci. The only definite allusion to these organisms, with respect to their utilization of dextrose, was found in the paper by Gordon (5). He states that the dextrose was not consumed by streptococci. Our own findings agree with those of Gordon.

2. *Sucrose.* Of the several sugars tested, sucrose seemed to be the most easily utilized. This is indicated in Tables *6* and *7* and in Series a, b, c in the section above on dextrose. Further evidence is presented in *Tables 8, 9,* and *10*. The data in *Table 11* show that sucrose may be detrimental. Reference to the literature reveals similar differences. Thus, Cohn (1) discussed the fact that bacteria grew just as well with-

out sucrose. Frankel and Pyle (2) state that, for several bacteria, addition of sucrose made the growth proceed more slowly.

3. *Glycerol.* The data in *Tables 12, 13, 14, 15, 16,* and *17* show that glycerol was often beneficial; in *Tables 18, 19* and *20,* that it was often without effect.

4. *Carbon compounds other than carbohydrates and glycerol: Citric, malic, lactic, and tartaric acids, and sodium lactate.* These, like glycerol, either induced no effect or were beneficial. The experimental data are recorded in *Tables 21, 22, 23,* and *24.* Further investigation showed that citric, lactic, malic, and tartaric acids were interchangeable for streptococcus metabolism. Details are given in *Tables 25* and *26.*

III. CONCLUSIONS

Among the carbon compounds studied, under the conditions specified, the non-carbohydrate substances were the most helpful for streptococcus nutrition.

Glycerol and the organic acids (citric, lactic, malic, tartaric) were more often beneficial than carbohydrates (dextrose, galactose, lactose, maltose, raffinose, sucrose) and mannite.

Of the carbohydrates, sucrose was usually the most beneficial, but it was also sometimes detrimental.

Dextrose was usually detrimental, and seldom beneficial in any degree.

IV. TABLES 1–26

TABLE 1

Dextrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION						RESULTS OF GROWTH					
									First transplant					
									Subcultures:					
		Cc. per 5 cc. before sterilization	$CaCl_2$	Dextrose	Glycerol	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	NaCl	1	2	3	4	5	6
579	5	0.01	0.01	1.0	1.0	0.2	0.1	0.5	+	0	0	0	0	0
580	5	0.01	0.01		1.0	0.2	0.1	0.5	3+	3+	3+	3+	3+	0

TABLE 2

Dextrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION										RESULTS OF GROWTH										
													First transplant						Second transplant				
													Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Dextrose	NH_4Cl	KNO_3	Histidine-HCl	KCl	$CaCl_2$	KH_2PO_4	$NaHCO_3$	Na_2SO_4	NaCl	1	2	3	4	5	6	1	2	3	4	5
124	6	0.02		0.1	0.05	0.1	0.0075	0.005	0.2	0.2	0.01	0.75	3+	3+	3+	2+	2+	2+	+	+	+	+	+
127	6	0.026	2.5	0.1	0.05	0.1	0.0075	0.005	0.2	0.2	0.01	0.75	0	0	0	0	0	0	0	0	0	0	0

TABLE 3

Dextrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION								RESULTS OF GROWTH										
											First transplant						Second transplant				
											Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Aspartic acid	Citric acid	Dextrose	Glycerol	$MgSO_4 \cdot 7H_2O$	K_2PO_4	K_2SO_4	NaCl	1	2	3	4	5	6	1	2	3	4	5
358	10	0.055	0.5	0.075	1.0	4.0	0.25	0.5	0.25	0.15	3+	0	0	0	0	0	0	0	0	0	0
359	10		0.5	0.075		4.0	0.25	0.5	0.25	0.15	4+	4+	4+	4+	3+	3+	0	+	5c	0	+
360	10	0.036	0.5		1.0	4.0	0.25	0.5	0.25	0.15	4+	4+	0	0	0	0	+	+	+	0	0
361[1]	10	0.03	0.5			4.0	0.25	0.5	0.25	0.15	4+	4+	3+	3+	2+	2+	+	+	4c	0	+

[1] Cc. of NaOH (2.123M) per 1 cc. after sterilization = 0.003.

TABLE 4

Dextrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION								RESULTS OF GROWTH						
												First transplant						
												Subcultures:						
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	$(NH_4)_2SO_4$	$Ca_3(PO_4)_2$	$FeSO_4 \cdot 7H_2O$	$MgSO_4 \cdot 7H_2O$	KCl	K_2HPO_4	Sulphur	Dextrose	1	2	3	4	5	6
84	13	0.005	0.002	0.2	1.0	0.001	0.05	0.05	0.1	1.0	0.01	0	0	0	0	0	0
85	13	0.009		0.2	1.0	0.001	0.05	0.05	0.1	1.0		3+	+	0	0	0	0

TABLE 5

Dextrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION							RESULTS OF GROWTH						
										First transplant						
										Subcultures:						
		Cc. per 5 cc. before sterilization	Alanine	$(NH_4)_2HPO_4$	Dextrose	$CaCl_2$	$MgSO_4 \cdot 7H_2O$]	K_2HPO_4	NaCl	1	2	3	4	5	6
583	5	0.04		1.0	1.0	0.01	0.2	0.1	0.5	3+	0	0	0	0	0
584	5	0.03		1.0		0.01	0.2	0.1	0.5	0	0	0	0	0	0
593	5	0.005	1.0		1.0	0.01	0.2	0.1	0.5	3+	0	0	0	0	0
594	5	0.005	1.0			0.01	0.2	0.1	0.5	0	0	0	0	0	0

TABLE 6

Carbohydrates

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION												RESULTS OF GROWTH										
															First transplant						Second transplant				
															Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Alanine	Aspartic acid	Dextrose	Galactose	Lactose	Maltose	Sucrose	$CaCl_2$	$FeSO_4 \cdot 7H_2O$	$MgSO_4 \cdot 7H_2O$	KH_2PO_4	Na_2CO_3	1	2	3	4	5	6	1	2	3	4	5
249	4		1.42						0.54	0.021	0.0003	0.024	0.14	0.21	3+	4+	3+	3+	3+	2+	+	0	0	0	0
251	4		1.42				0.54			0.021	0.0003	0.024	0.14	0.21	3+	2+	2+	+	+	0	0	0	0	0	0
252	4		1.42					0.54		0.021	0.0003	0.024	0.14	0.21	4+	3+	3+	2+	2+	0	0	3c	0	0	0
250	4		1.42			0.54				0.021	0.0003	0.024	0.14	0.21	3+	2+	0	0	0	0	0	0	0	0	0
247	4		1.42		0.54					0.021	0.0003	0.024	0.14	0.21	3+	0	0	0	0	0	0	0	0	0	0
237[1]	4	0.13		1.2					0.54	0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	3+	3+	3+	+	0	0	0	+
238	4	0.155		1.2		0.54				0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	3+	2+	0	0	+	4c	0	2c
235	4	0.15		1.2	0.54					0.021	0.0003	0.024	0.14	0.21	3+	3+	2+	+	+	0	+	1c	0	0	0
240	4	0.148		1.2				0.54		0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	0	0	0	+	0	0	0	0
239	4	0.148		1.2			0.54			0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	+	+	0	+	0	0	0	0

[1] Cc. of NaOH (2.123M) per 1 cc. after sterilization = 0.004.

TABLE 7

Carbohydrates

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION										RESULTS OF GROWTH					
														First transplant					
														Subcultures:					
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	Dextrose	Lactose	Maltose	Mannite	Raffinose	Sucrose	NH_4Cl	Glycerol	$MgSO_4 \cdot 7H_2O$	KH_2PO_4	1	2	3	4	5	6
395	10	0.09							1.0	0.2	1.5	0.25	0.5	+	+	+	+	0	0
400	10	0.09			1.0					0.2	1.5	0.25	0.5	3+	2+	0	0	0	0
410	10	0.1				1.0				0.2	1.5	0.25	0.5	4+	+	0	0	0	0
405	10	0.12						1.0		0.2	1.5	0.25	0.5	+	0	0	0	0	0
384	10	0.09	0.002	1.0						0.2	1.5	0.25	0.5	3c	0	0	0	0	0
390[1]	10	0.08	0.005				1.0			0.2	1.5	0.25	0.5	0	0	0	0	0	0

[1] In medium 390 mannite was substituted for mannose, which was used by Proskauer and Beck.

TABLE 8

Sucrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION					RESULTS OF GROWTH						
								First transplant						
								Subcultures:						
		Cc. per 5 cc. before sterilization	$(NH_4)_2CO_3 \cdot H_2O$	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	Sucrose	1	2	3	4	5	6
15a	0[1]	0.075	0.5	0.0025	0.5	0.5		0	0	0	0	0	0
15m	0	0.075	0.5	0.0025	0.5	0.5	3.0	2+	+	0	0	0	0

[1] "0" in the column under "Bibliography," here and in subsequent tables, indicates that the medium has been devised by the authors.

TABLE 9

Sucrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION				RESULTS OF GROWTH										
							First transplant						Second transplant				
							Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Aspartic acid	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	Sucrose	1	2	3	4	5	6	1	2	3	4	5
27a	0	0.04	0.2	0.0036	0.1		2+	2+	2+	0	0	0	+	1c	0	0	0
27m	0	0.04	0.2	0.0036	0.1	3.0	2+	2+	2+	2+	+	0	+	0	0	0	0

TABLE 10

Sucrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION						RESULTS OF GROWTH					
									First transplant					
									Subcultures:					
		Cc. per 5 cc. before sterilization	$MgCl_2$	KNO_3	NaCl	$Na_2HPO_4 \cdot 12H_2O$	$Na_2S_2O_3 \cdot 5H_2O$	Sucrose	1	2	3	4	5	6
686	9	0.03	0.25	0.1	3.0	0.05	0.2		0	0	0	0	0	0
687	9	0.03	0.25	0.1	3.0	0.05	0.2	0.5	3+	3+	2+	0	0	0

TABLE 11

Sucrose

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION							RESULTS OF GROWTH					
											First transplant					
											Subcultures:					
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	Aspartic acid	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	Malic acid	Na_2CO_3	$Na_2HPO_4 \cdot 12H_2O$	Sucrose	1	2	3	4	5	6
510	3	0.27		1.0	0.001	0.2	0.7	0.25	0.2		3+	+	0	0	0	0
511	3	0.31	0.004	1.0	0.001	0.2	0.7	0.25	0.2	1.5	0	0	0	0	0	0

TABLE 12

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION									RESULTS OF GROWTH					
												First transplant					
												Subcultures:					
		Cc. per 5 cc. before sterilization	Glycerol	Histidine HCl	KCl	$CaCl_2$	KH_2PO_4	$NaHCO_3$	Na_2SO_4	NaCl	KNO_3	1	2	3	4	5	6
119	6	0.024		0.1	0.0075	0.005	0.2	0.2	0.01	0.75		+	0	0	0	0	0
121	6	0.022	2.0	0.1	0.0075	0.005	0.2	0.2	0.01	0.75		3+	3+	3+	3+	+	+
123	6	0.019		0.1	0.0075	0.005	0.2	0.2	0.01	0.75	0.05	0	0	0	0	0	0
125	6	0.019	2.0	0.1	0.0075	0.005	0.2	0.2	0.01	0.75	0.05	2+	2+	3+	2+	2+	0

TABLE 13

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION								RESULTS OF GROWTH					
											First transplant					
											Subcultures:					
		Cc. per 5 cc. before sterilization	NH_4Cl	$(NH_4)_2SO_4$	Citric acid	Mg citrate	$MgSO_4 \cdot 7H_2O$	KH_2PO_4	Mannite	Sucrose	Glycerol	1	2	3	4	5	6
398	10	0.11		0.2		0.25		0.5		1.0	1.5	3+	2+	2+	2+	+	+
399	10	0.03		0.2		0.25		0.5		1.0		2+	2+	0	0	0	0
429	10	0.12	0.2		0.075		0.25	0.5	0.6		1.5	2+	2+	2+	+	+	+
430	10	0.11	0.2		0.075		0.25	0.5	0.6			0	0	0	0	0	0

TABLE 14

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION									RESULTS OF GROWTH										
												First transplant						Second transplant				
												Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	NH_4Cl	Glycerol	Histidine-HCl	KCl	$CaCl_2$	KH_2PO_4	$NaHCO_3$	Na_2SO_4	NaCl	1	2	3	4	5	6	1	2	3	4	5
120	6	0.02	0.1		0.1	0.0075	0.005	0.2	0.2	0.01	0.75	4+	4+	+	2+	+	4c	+	+	+	3c	4c
122	6	0.019	0.1	2.0	0.1	0.0075	0.005	0.2	0.2	0.01	0.75	3+	4+	4+	4+	4+	4+	+	4c	3c	2c	3c

TABLE 15

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION				RESULTS OF GROWTH										
							First transplant						Second transplant				
							Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Glycerol	Aspartic acid	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	1	2	3	4	5	6	1	2	3	4	5
27a	0	0.04		0.2	0.0036	0.1	2+	2+	2+	0	0	0	+	1c	0	0	0
27t	0	0.031	2.0	0.2	0.0036	0.1	2+	2+	2+	2+	2+	+	+	0	0	0	1c

TABLE 16

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION									RESULTS OF GROWTH					
												First transplant					
												Subcultures:					
		Cc. per 5 cc. before sterilization	$(NH_4)_2SO_4$	$(NH_4)_2$ tartrate	Glycerol	Mg_3 citrate	$MgSO_4 \cdot 7H_2O$	KH_2PO_4	Lactose	Maltose	Mannite	1	2	3	4	5	6
403	10	0.13	0.2		1.5	0.25		0.5	1.0			3+	2+	+	0	0	0
404	10	0.17	0.2			0.25		0.5	1.0			3+	0	0	0	0	0
413	10	0.13	0.2		1.5	0.25		0.5		1.0		3+	2+	+	+	+	0
414	10	0.15	0.2			0.25		0.5		1.0		3+	2c	0	0	0	0
425	10	0.13		0.2	1.5		0.25	0.5			0.6	3+	3+	3+	3+	2+	2+
426	10	0.14		0.2			0.25	0.5			0.6	3+	3+	3+	3+	2+	2+
427	10	0.11	0.2		1.5	0.2		0.5			0.6	3+	+	0	0	0	0
428	10	0.10	0.2			0.2		0.5			0.6	+	2c	0	0	0	0

TABLE 17

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION				RESULTS OF GROWTH					
							First transplant					
							Subcultures:					
		Cc. per 5 cc. before sterilization	Glycerol	Aspartic acid	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	1	2	3	4	5	6
175	12	0.174		1.0	0.02	0.1	3+	2+	+	5c	0	0
180	12	0.173	2.0	1.0	0.02	0.1	4+	3+	3+	3+	2+	0

TABLE 18

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION					RESULTS OF GROWTH					
								First transplant					
								Subcultures:					
		Cc. per 5 cc. before sterilization	Glycerol	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	$(NH_4)_2CO_3 \cdot H_2O$	1.	2	3	4	5	6
15a	0	0.075		0.0025	0.5	0.5	0.5	0	0	0	0	0	0
15t	0	0.076	2.0	0.0025	0.5	0.5	0.5	0	0	0	0	0	0

TABLE 19

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION						RESULTS OF GROWTH					
									First transplant					
									Subcultures:					
		Cc. per 5 cc. before sterilization	Glycerol	$MgCl_2$	KNO_3	NaCl	$Na_2HPO_4 \cdot 12H_2O$	$Na_2S_2O_3 \cdot 5H_2O$	1	2	3	4	5	6
686	9	0.03		0.25	0.1	3.0	0.05	0.2	0	0	0	0	0	0
688	9	0.03	0.5	0.25	0.1	3.0	0.05	0.2	0	0	0	0	0	0

TABLE 20

Glycerol

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION									RESULTS OF GROWTH										
												First transplant						Second transplant				
												Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	$(NH_4)_2SO_4$	$(NH_4)_2$ tartrate	Glycerol	Mg_3 citrate$_2$	$MgSO_4 \cdot 7H_2O$	KH_2PO_4	Lactose	Mannite	Raffinose	Sucrose	1	2	3	4	5	6	1	2	3	4	5
391[1]	10	0.16		0.2	1.5		0.25	0.5		1.0			3+	2+	2+	2+	0	0	0	0	0	0	0
392[1]	10	0.14		0.2			0.25	0.5		1.0			3+	3+	3+	2+	0	0	0	+	0	0	0
393[1]	10	0.12	0.2		1.5	0.25		0.5		1.0			3+	2+	2+	2+	+	0	5c	0	0	0	0
394[1]	10	0.12	0.2			0.25		0.5		1.0			3+	3+	3+	3+	+	0	3c	0	0	0	0
396	10	0.13		0.2	1.5		0.25	0.5				1.0	3+	3+	3+	3+	3+	0	+	+	+	0	0
397	10	0.13		0.2			0.25	0.5				1.0	3+	3+	3+	3+	2+	0	+	4c	2c	+	0
401	10	0.15		0.2	1.5		0.25	0.5	1.0				+	0	0	0	0	0	0	0	0	0	0
402	10	0.13		0.2			0.25	0.5	1.0				2+	+	0	0	0	0	0	0	0	0	0
406	10	0.14		0.2	1.5		0.25	0.5			1.0		2+	2+	2+	2+	2+	2+	3c	3c	0	0	0
407	10	0.17		0.2			0.25	0.5			1.0		3+	3+	2+	2+	2+	2+	2c	0	0	0	0
408	10	0.12	0.2		1.5	0.25		0.5			1.0		3+	+	2+	2+	2+	3+	0	0	0	0	0
409	10	0.10	0.2			0.25		0.5			1.0		2+	2+	+	2+	2+	2+	2c	0	0	0	0

[1] In media 391–394 mannite was substituted for mannose, which was used by Proskauer and Beck.

TABLE 21

Citric acid

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION				RESULTS OF GROWTH											
							First transplant						Second transplant					
							Subcultures:						Subcultures:					
		Cc. per 5 cc. before sterilization	Citric acid	Aspartic acid	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	1	2	3	4	5	6	1	2	3	4	5	
27a	0	0.04		0.2	0.0036	0.1	2+	2+	2+	0	0	0	+	1c	0	0	0	
27s	0	0.253	1.0	0.2	0.0036	0.1	2+	2+	2+	+	0	0	0	0	0	0	0	

TABLE 22

Citric acid

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION								RESULTS OF GROWTH					
											First transplant					
											Subcultures:					
		Cc. per 5 cc. before sterilization	NH_4Cl	Citric acid	Dextrose	Glycerol	$MgSO_4 \cdot 7H_2O$	KH_2PO_4	K_2SO_4	NaCl	1	2	3	4	5	6
382	10	0.09	0.2	0.075	1.0	1.5	0.25	0.5	0.25	0.12	3+	0	0	0	0	0
383	10	0.117	0.2		1.0	1.5	0.25	0.5	0.25	0.12	3+	0	0	0	0	0

TABLE 23

Citric acid and sodium lactate

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION						RESULTS OF GROWTH					
									First transplant					
									Subcultures:					
		Cc. per 5 cc. before sterilization	Citric acid	Na lactate	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	$(NH_4)_2CO_3 \cdot H_2O$	1	2	3	4	5	6
15a	0	0.075			0.0025	0.5	0.5	0.5	0	0	0	0	0	0
15o	0	0.07		0.5	0.0025	0.5	0.5	0.5	2+	+	0	0	0	0
15s	0	0.155	3.0		0.0025	0.5	0.5	0.5	2+	2+	2+	2+	2+	2+

TABLE 24

Sodium lactate

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION					RESULTS OF GROWTH										
								First transplant						Second transplant				
								Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Na lactate	Aspartic acid	$MgSO_4 \cdot 7H_2O$	KCl	K_2HPO_4	1	2	3	4	5	6	1	2	3	4	5
27j	0	0.039		0.2	0.0036	0.05	0.1	3+	3+	0	0	0	0	0	0	0	0	0
27jV	0	0.039	0.5	0.2	0.0036	0.05	0.1	2+	3+	3+	2+	0	0	+	0	3c	0	0

TABLE 25

Organic acids

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION									RESULTS OF GROWTH					
													First transplant					
													Subcultures:					
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	Citric acid	Lactic acid	Malic acid	Tartaric acid	Aspartic acid	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	Na_2CO_3	$Na_2HPO_4 \cdot 12H_2O$	1	2	3	4	5	6
461	8	0.263	0.006			0.7		1.0	0.001	0.04	0.25	0.2	3+	2+	0	0	0	0
515	8	0.21	0.02		0.7			1.0	0.001	0.04	0.25	0.2	3+	3+	2+	+	0	0
516	8	0.24	0.018				0.7	1.0	0.001	0.04	0.25	0.2	3+	2+	2+	+	0	0
517	8	0.3	0.01	0.7				1.0	0.001	0.04	0.25	0.2	3+	2+	2+	0	0	0

TABLE 26

Organic acids

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2 123M		PERCENTAGE COMPOSITION								RESULTS OF GROWTH					
												First transplant					
												Subcultures:					
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	Aspartic acid	Glycerol	$MgSO_4 \cdot 7H_2O$	K_2PO_4	Citric acid	Lactic acid	Malic acid	Tartaric acid	1	2	3	4	5	6
365	10	0.04	0.006	0.5	1.0	0.25	0.5	0.075				3+	3+	3+	4c	0	4c
366	10	0.06		0.5	1.0	0.25	0.5		0.1			3+	3+	3+	2+	3+	0
367	10	0.09		0.5	1.0	0.25	0.5				0.075	3+	3+	3+	2+	+	0
368	10	0.04	0.008	0.5	1.0	0.25	0.5			0.075		3+	3+	3+	+	0	0

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ORAL MICROÖRGANISMS OF THE DOG[1]

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Before the dawn of historic time man betook to himself, from the animals of the forests, such as were found to meet his needs. According to evidence thus far revealed, it was late in the Stone Age, as late as the earliest Neolithic period, that domestication became established. The first animal that shared the household of man appears to have been the dog. Before the period of the stone-graves and the flat-poled ax, remains of the dog were common in the Danish kitchen-middens. In all subsequent Neolithic and Bronze Age deposits, its presence was in evidence. At the end of the Bronze Age several species existed. During the Viking period, dogs were found interred, together with a chieftain, in the Gokstad burial ship. Man's association with the dog, according to moderate estimates, dates back to about 1500 years B. C. Over three thousand years of close commensalism should be sufficient, if the excellent opportunities for interchange of existing parasitic or symbiotic flora or fauna be considered, to afford opportunity for adaptations of microbes from either one to the other of the hosts.

It is well known that the human mouth harbors a microbial film. It is obvious that some dogs, through their subsistence on table scraps, such as gnawed bones and the like, ingest microörganisms from the human mouth almost daily. It is equally certain that Fido, when he kisses his mistress or licks the hands of children, or barks in proximity to his masters, thereby transmits representatives from his own film. Such possibilities clearly indicate the importance of a systematic study of the film organisms.

[1] Work supported, in part, by a grant from the Research Commission of the American Dental Association. Read at a session of the Seventh Annual Meeting of the International Association for Dental Research, Northwestern University Dental School, Chicago, Ill., March 23–24, 1929. See abstract: *Journal of Dental Research*, 1929, ix, p. 288.

Reasons for the tolerance of microbial films by their hosts are not yet clear. From the small number of varieties found to persist in the oral cavity, notwithstanding the enormous intake, it must be inferred that these are specifically adapted to this environment. The varieties growing in the human mouth, for example, are so few, that it has been possible, through the research done since Robin's and Leber and Rottenstein's time, to designate less than ten forms that alone compose the bulk of the *materia* found upon the teeth. F. Vicentini, of Chieta, Italy, the discoverer of the Leptothrix racemosa, was the first to study the film upon dog's teeth. It was at the suggestion of Dr. V. A. Latham, of Chicago, a former co-worker of F. Vicentini, that the observations carried on in this laboratory were extended to the oral flora of the dog. As many of the film organisms rarely, perhaps never, respond to attempts at their cultivation on artificial media, morphological studies of the organisms found in smears were undertaken. At the 1928 meeting of the International Association for Dental Research I reported on some progress in this work.[2]

Before entering into a brief description of some of the forms, a few generalities will characterize the impressions gained from an examination of smears made from fifteen dogs. Two to three smears, each containing film from both the crown and subgingival space, were made from each animal. The smears, on the whole, display much similarity with those made from human material, although they differ in various details. A few distinctive characters may be expressed as follows.

(1) The film in the mouth of man far exceeds in amount the growth exhibited by the dog. The dog's mouth is the cleaner of the two.

(2) Spirochaetes are invariably found in both cases, the dog harboring, besides the Spir. buccalis type, a variety that is lacking in the human.

(3) Spirillum sputigenum and fusiformed organisms are found in both man and dog, the human forms being larger, more chromophilic, and as a rule much more numerous.

(4) Both hosts exhibit the thread forms generally known as leptothrix filaments. Of these the dog harbors two morphological varieties not found in the mouth of man: one of great dimensions, and another

[2] Beust: *Journal of Dental Research*, 1928, viii, p. 439.

displaying a flexibility that deviates from any of the human forms. I am not yet convinced that other thread forms found in the dog's mouth are identical with the human organisms.

(5) Cocci are fairly constant in both cases. Dogs have more diplococci, especially of the lancet-shaped variety. Chain-forming cocci I have never found in the smears of man or dog.

The early students of the oral film commented on the observation that many of the mouth microörganisms appeared to grow from the clumps of *materia* commonly present in mouth smears. Such clumps were called the matrix of the organisms. In 1892 Vicentini, in the discovery of Leptothrix racemosa, laid the foundation for a newer conception of the word matrix. Although he never, to my knowledge, made use of that term himself, he showed through his work that the spores of his organism divided and subdivided until finally there appeared that which in the smears could be pronounced a clump, or matrix, from which various organisms were seen to grow. Vicentini claimed that such a propagation also occurred in the mouth of the dog. He speaks of fruit heads, meaning the spore forming stages of his racemosa, occurring in the dogs film. Personally, in the fifteen dogs examined, I have not yet found an undoubted specimen of the Lep. racemosa. Perhaps further search will reveal its presence, but these forms are certainly less plentiful than in the human mouth. Matrix forms, on the other hand, were readily found.

Fig. 1 displays a matrix form with flexible thread-like conidia of extreme length. Dismembered fragments of these bodies may easily be mistaken for spirochaetes. The core of the matrix consists of countless slender, spindle-shaped bacilli, which surround a central stem.

Fig. 2 shows a camera-lucida drawing of an organism belonging to the variety shown in *fig. 1*. The threads growing from the core differ strikingly, in their marked undulations, from the threads of the human mouth. These filaments may reach the length of nearly 150 microns. Some are found to be regularly septated while in others the septation is lacking.

Figs. 3 and *4* exhibit a form in which only the core, consisting of spindle-shaped bacilli with central axial thread, is present. The bacilli composing these cores are more delicate, less sturdy, and ordinarily less chromophilic, than those found in human material.

FIG. 1. PHOTOMICROGRAPH SHOWING CORE WITH FILAMENTS APPARENTLY EMANATING THEREFROM

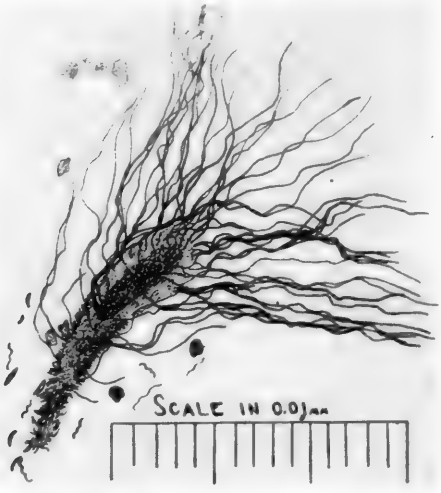

FIG. 2. CAMERA LUCIDA DRAWING, WITH SCALE SHOWING THE LENGTH OF THE FILAMENTS

Fig. 5 shows further specimens. The larger thread is a part of a Leptothrix gigantea, the filaments of which often reach a length of over 500 microns. Stained with iron-haematoxylin, these organisms

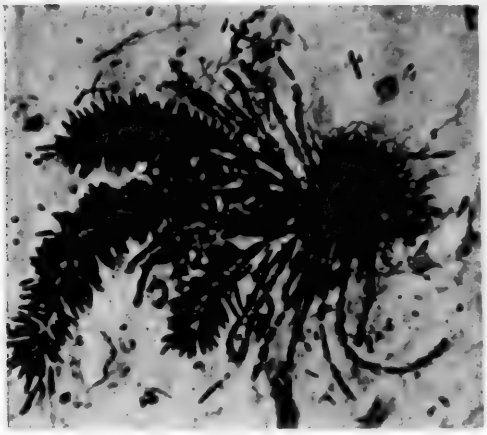

FIG. 3. PHOTOMICROGRAPH OF THE ORGANISM SHOWN IN *fig. 4*

FIG. 4. CAMERA LUCIDA DRAWING OF A MATRIX-FORMING ORGANISM

The threads, together with the fusiform spore-bodies thereon, are regarded by the author as organic units.

are generally found to be regularly septated; unseptated specimens however are also found. Some of the threads are seen to be devoid of protoplasm (*fig. 6*). In such cases the septa between the cell walls

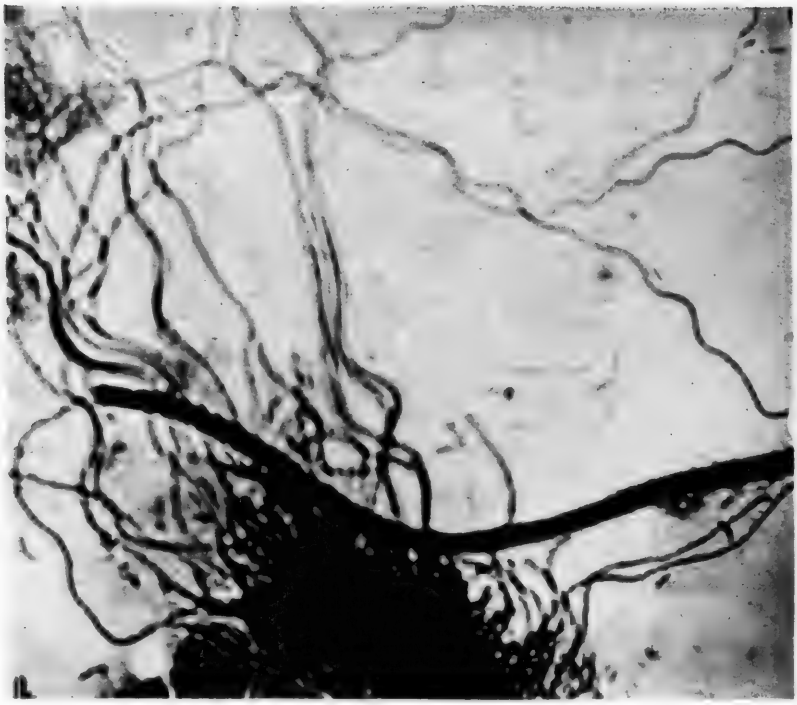

FIG. 5. SMEAR SHOWING UNDULATING FILAMENTS AND A PART OF A LEPTOTHRIX GIGANTEA (THICK)

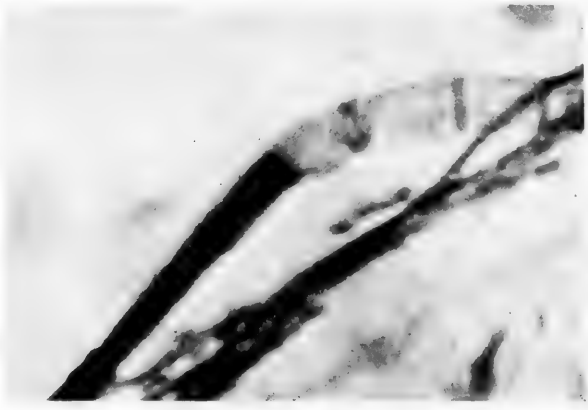

FIG. 6. A FEW SEGMENTS OF LEPTOTHRIX GIGANTEA SHOWING DIVIDING CELL WALLS IN THE EMPTY SHEATH

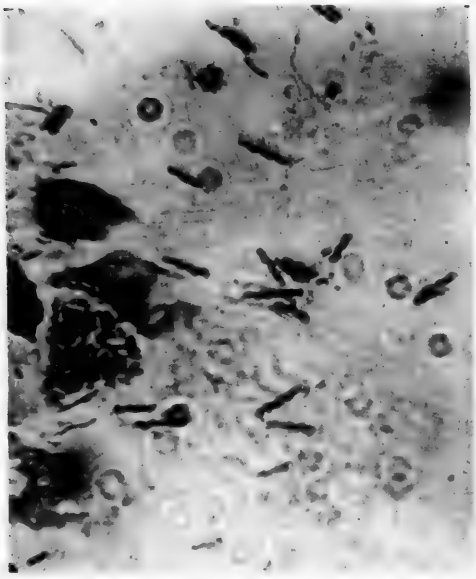

FIG. 7 FIG. 8

FIG. 7. AN ABERRANT FORM OF SPIROCHAETE

FIG. 8. CAMERA LUCIDA DRAWING OF ABERRANT SPIROCHAETE FORMS (THICK), WITH THE SPIROCHAETA BUCCALIS (THIN) FOR COMPARISON

The scale is in microns

FIG. 9. PROTOZOAN-LIKE ORGANISMS, SHOWING THEIR MODE OF OCCURRENCE IN CELLS, OR IN SEMI-TRANSPARENT FILMS

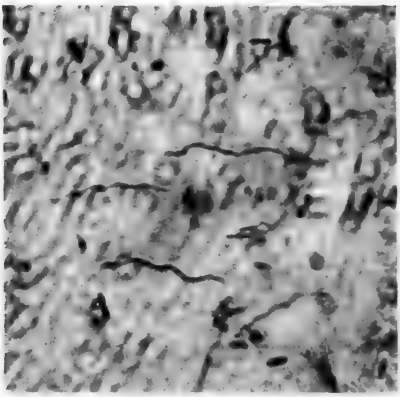

FIG. 10. SPIROCHAETES OF THE SP. BUCCALIS TYPE

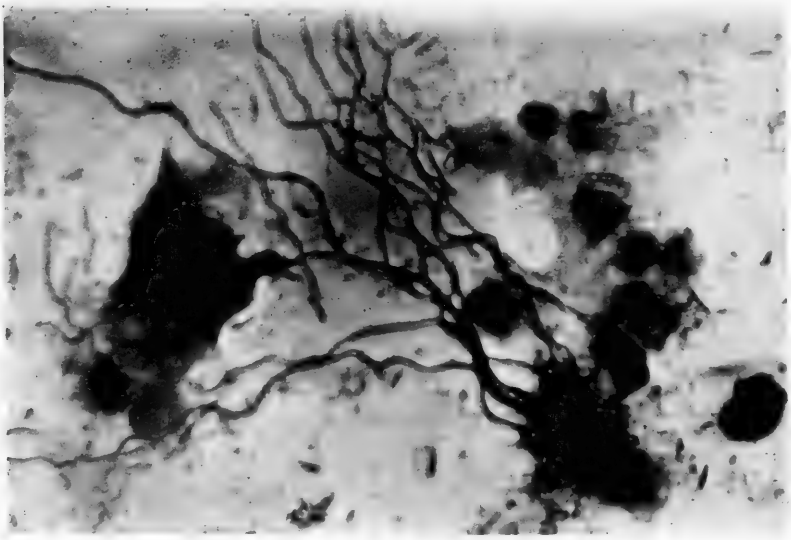

FIG. 11. FRAGMENT OF A MATRIX-FORMING ORGANISM AS COMMONLY FOUND IN PROPERLY PREPARED SMEARS

are plainly visible. Such septa were shown by the writer to occur in the leptothrix threads of the human mouth also.[3] The presence of these empty cell walls may indicate the occurrence of zoospore, or gamete, formation.

A spirochaete which differs from any form found in the human mouth is represented in *figs. 7 and 8*. This is a short thick form found in practically all of thirty smears. It is about six microns in length and usually exhibits three or four shallow windings. It is always straight, and is apparently rigid. A feature of this organism is its diameter, which far exceeds that of the common buccal types. Several of the latter are shown in the drawing for comparison. The thick form measures about 0.7 micron in diameter. In well differentiated smears these spirochaetes display course granulations in their protoplasm.

Another organism, which has never appeared in any of my human material, is shown in *fig. 9*. This may possibly be a protozoan. They are often found embedded in epithelial cells or in transparent films. They average about 10 microns in length and 4 microns in diameter. The transverse septa, shown in the incomplete specimen at the left in *fig. 9*, in some cases seem to reach 16 in number. In many organisms 14 can easily be counted. Further study will be necessary to fully describe these forms.

Owing to the scarcity of contributions of this nature to our literature, I have refrained from naming any of these forms. While a reasonable degree of certainty exists that the matrix-forming organisms have never before been described, it is not altogether impossible that earlier descriptions of the others shown have escaped my attention.

I hope this report will stimulate others to begin investigations along these lines.

[3] Beust: *Dental Items of Interest*, 1909, xxxi, p. 99.

MICRO-CINEMATOGRAPHY IN MEDICAL RESEARCH[1]

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The motion picture is an extremely valuable tool for the demonstration of facts. An old proverb reads: "One picture is worth more than many thousand words." The usefulness of the motion picture is especially obvious in medical science. Many records are made of surgical operations, of presentations of cases, etc. Today all the institutions of learning know the importance of the motion picture as a means of demonstration. By its domination of time, the motion picture is also a great help to research workers, especially where the human eye has its limits. We are chiefly concerned with the motion picture, especially the microscopic motion picture, as a means for investigation.

The use of motion pictures for the analysis of movements of various subjects, especially men and animals, is as old as cinematography itself. I might call your attention to the experiments of Muybridge in America, and of Marey in France, about the year 1890. As a matter of fact we can safely say that the motion picture originated in the biological laboratory. Muybridge used a whole battery of still cameras, to be released by strings, which were placed in a row. In this way he took consecutive pictures of walking men and animals in order to study the various phases of their movements. Marey used for his experiments only one camera of the ordinary type, but placed in front of the plate a revolving shutter with slots in it, and made a number of exposures of the moving subject on the same plate. He studied, for example, the movements of a rabbit or a cat which had been dropped from a certain height. He also extended his researches to the study of flying birds by using a kind of photographic gun, and obtained consecutive pictures representing the progressive movements.

[1] Read at a meeting of the First District Dental Society of the State of New York, New York Academy of Medicine, February 4, 1929.

These experiments, conducted about forty years ago, were made because the human eye cannot perceive motions that are above or beyond a certain speed limit.

Motion picture science has been developed considerably. We can see today, in the moving-picture houses, films taken with the slow motion camera and are able to observe step by step the movements of tennis players, fighters, swimmers, race horses, and many other things. Not long ago motion pictures, exhibited by a Japanese professor, were taken at a rate of several thousand per second. They showed, for instance, how a bullet from a shot-gun penetrated an electric bulb. Here the motion picture is used as a time stretcher, but it can also be employed to concentrate the time (for speeding up). The device for this purpose is called the time-lapse camera.

The normal rate of projection, as we know, is sixteen pictures per second. If the taking-speed is more than sixteen, then the projected picture is slowed down. Any motion taken at a rate of less than sixteen per second is accelerated on the screen. Thus we are able actually to see the growing of plants, the opening of flowers, and many other phenomena. We are able to observe in a few seconds or minutes what actually took place in many hours or perhaps days. This method of time stretching or acceleration can also be applied in the microscopic field, and surpasses in fact the best trained observer. The accelerated micro-cinema film has become a great help to medical and biological investigators. We have to give credit to a few men who used the micro-cinema some eighteen or twenty years ago, namely Chevroton and Comandon in France, and Neuhaus and Scheffer in Germany. Especially Comandon became well-known through his presentations before the French Academy of Sciences.

Micro-cinematography has been derived from microphotography, and is theoretically built up on the same principles, the only differences being that the still camera is replaced by the moving-picture camera, with its driving and timing mechanism; and, of course, the subject is living and moving instead of dead and stained. One encounters a number of difficulties that do not occur in still microphotography. The source of light has to be more powerful, especially with darkfield illumination. The preparation is in most cases very delicate, and is sensitive to excessive light and heat. The slightest

vibration will be magnified in the microscope. A living preparation is likely to go out of focus, and a control of focus is frequently necessary. The field of vision is only the size of a motion-picture frame, being only about one inch broad. In taking the pictures one has to perform a number of manipulations at practically the same time.

The micro-cinema apparatus, as it is now used, is composed of a number of special devices required for the different subjects. Today there are a few focussing instruments on the market to be used for microscopic motion pictures. These are oculars combined with a so-called beam splitter. For more serious work, however, and excellent results, a complete equipment is essential. At the end, the better the equipment is, the less material is wasted. The apparatus used for micro-cinema work is somewhat specialized for the different subjects to be taken. The principal parts are the microscope, with the source of light and optical bench on one table; the camera, with the driving mechanism, timer, and revolving shutter on another table. These two sets are entirely separate from each other in order to prevent the transmission of vibrations of motor and camera. For the same reason the motor is placed as far from the apparatus as possible. In working with high magnifications the prevention of vibration becomes a difficult problem and has to be reckoned with by selecting a proper room as the laboratory. *Fig. 1* represents the apparatus used at the Rockefeller Institute for Medical Research, since 1923, for taking film records of living animal-tissue cells.

Dr. Alexis Carrel, as is known, was the first one who succeeded in keeping fragments of various human and animal tissues alive outside of the body for a long period of time. The fragments removed from the animal are put into flasks, and a proper nutrient liquid consisting of plasma and embryonic juice is substituted. When kept in an incubator at body temperature and free from bacterial infection, these cultures thrive and multiply continuously for an unlimited time. A culture of heart tissue, for instance, taken by Dr. Carrel from a chick embryo seventeen years ago (1912), is today still alive and multiplying. Dr. Carrel claims that tissue is immortal. The cultures are washed and refilled with nourishment from time to time, by injecting the nutrient fluid. Both these manipulations are done by means of a pipette. Then the neck is flamed in order to protect the inside from

bacterial contamination. The flasks cannot be used for micro-cinema work. It has been found that a perforated slide, 1 x 3 inches in size, is more suitable from the optical point of view. The culture is sealed in, and a drop of nutrient liquid hangs between the cover-glasses. The space around the drop is air. In this the culture can live for from 12 to 24 hours.

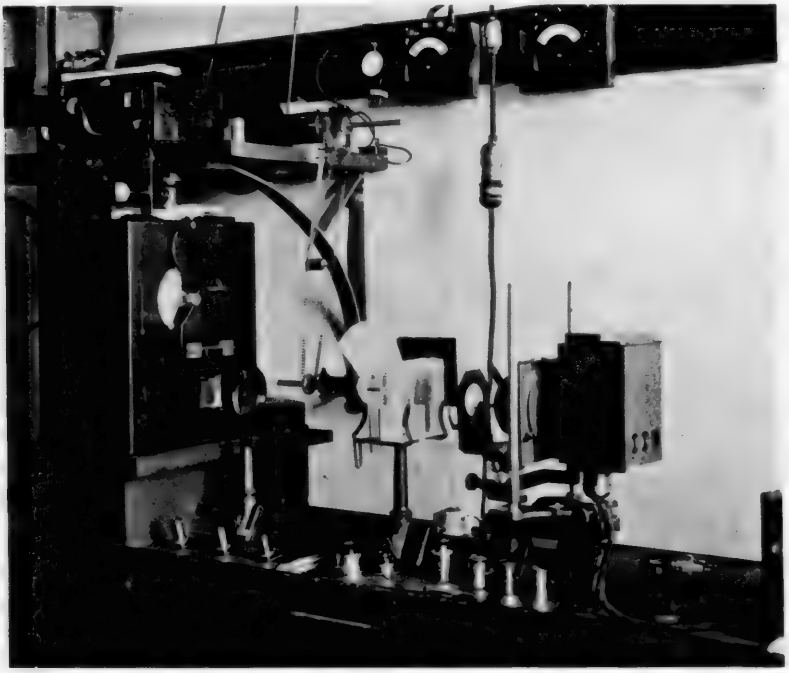

FIG. 1. Micro-cinema apparatus, Model 1923, at the Rockefeller Institute for Medical Research, for film records of living cells.

The film shows to us these cells in a striking realism. Cells of fibroblast, for instance, taken under low power, crawl across the screen and form a dense tissue which finally covers the whole field. Under medium power, single cells can be clearly observed as they move forward, with an active to-and-fro movement of the cell granules. Under high power, single cells and even parts of cells can be focused. The

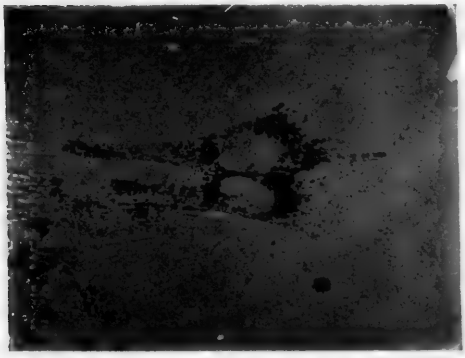

FIG. 2. Two single cells of chick embryo fibroblasts. Cell nucleus, nucleoli, granules, vesicles, well shaped cell boundaries. High magnification.

FIG. 3. Two cells of fibroblasts from chick embryo. The nucleus with its two nucleoli can be seen clearly. Dark-field illumination.

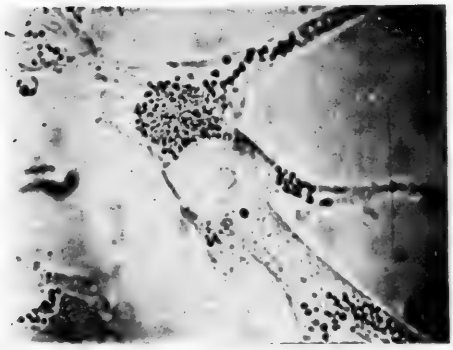

FIG. 4. One cell of rat sarcoma fibroblast. Nucleus with nucleolus, cell granules, mitochondria. Very high magnification.

FIG. 5. White blood cells, just removed from the blood. Dark field.

action of single granules and mitochondria, which are less than 1μ in diameter, can be closely studied. Every part of these cells appears to be highly active, especially around the centriole. Experiments

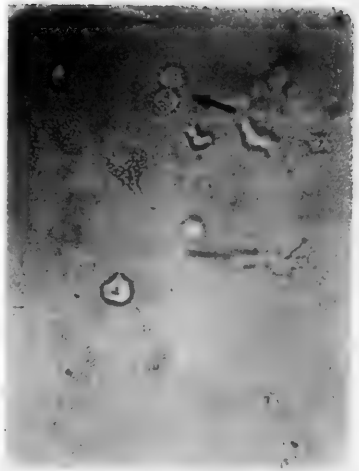

FIG. 6

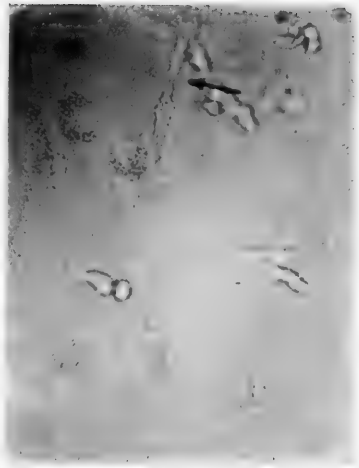

FIG. 7

FIG. 8

FIG. 6. Cell division of a white blood corpuscle. See also *fig. 7*.
FIG. 7. Cell division of a white blood corpuscle. Compare with *fig. 8*.
FIG. 8. After cell division, one of the divided cells (left one) is taken up by another cell.

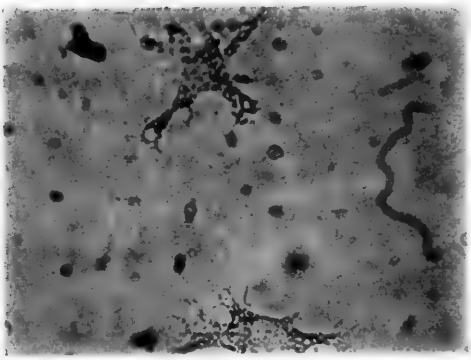

FIG. 9. Formation of giant cells from guinea pig. See also *fig. 10.*

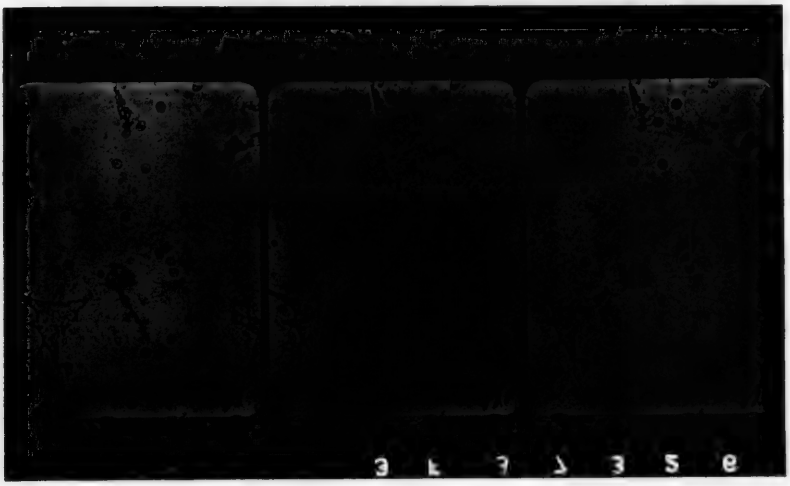

FIG. 10. Formation of giant cells from guinea pig. Phagocytosis of small lymphocyte.

have been made with cells of various other tissues, normal or malignant; cells of the blood, etc.; and occasionally in bacteriology and chemistry. Thus it has been found that the micro-cinema can be of great help in nearly all sciences in which microscopical observations on living material are made.

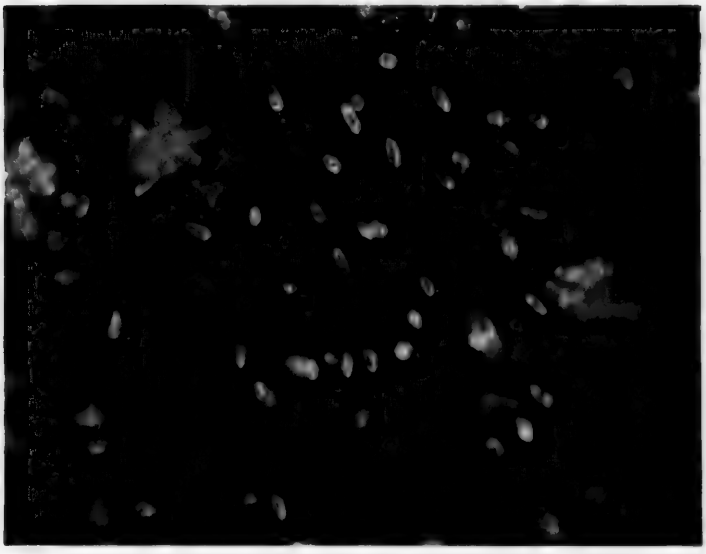

FIG. 11. Culture of Herpetomanos lygaeorum, a flagellate. Taken with the late Dr. Hideyo Noguchi, Division of Bacteriology.

FIG. 12. Culture of Bacterium coli. Within six hours the field was entirely covered by the growth from a few bacteria originally in the field. Experiment made with Dr. Bronfenbrenner.

The accompanying illustrations (*figs. 2–12*),[2] which were made from the original film negatives, give evidence of the usefulness of the micro-cinema in the various medical branches. *Figs. 2* to *10* are from the film, "The structure of living cells," taken in collaboration with Dr. Alexis Carrel and Dr. H. H. Ebeling. *Fig. 11* is from the film, "Comparative studies of herpetomonads and leishmanias," taken in collaboration with the late Dr. Hideyo Noguchi. *Fig. 12* is from the film, "The action of bacteriophage on bacterium coli," taken in collaboration with Dr. J. J. Bronfenbrenner.

Micro-cinema has opened an immense field in medical and biological research, and in all natural sciences. Instead of seeing in the microscope only the apparently motionless images of the specimens, as we look at the illustrations of a book, we are now able to observe upon the screen how these small cells live and behave. It is possible to study the action of malignant cells on normal cells, and also the action of radiations (x-ray or radium) or chemicals on cells, which may help to clarify the cancer problem.

[2] *Fig. 10* shows three successive views in their sequence on the film. Similar groups were presented by the author for all of the remaining illustrations excepting *fig. 1*, but in each of these cases only one is reproduced here. The differences were not greater than those shown in *fig. 10*, which is given in its entirety, although unavoidably reduced in size (compare with *figs. 6–9*), as a representative of the entire series.—[*Ed.*]

A SADDLE, PHYSIOLOGICAL IN CONSTRUCTION AND DESIGN, FOR PRECISION ATTACHMENTS[1]

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I. CHART ONE

Definition.—Saddles are vehicles that fit the alveolar ridge for the restoration of lost teeth and their alveolar processes, and conform to certain physiological, esthetic, and mechanical requirements. "Precision attachments" as here employed are such bridge-abutment attachments as resemble the inlay bucco-lingual attachments. The object of this clinic is an additional adjustment of the saddle to its ridge.

Point of view.—In analysing the various factors involved in saddle construction, the ridge upon which it is to be seated is of prime consideration. The physiological and mechanical aspects of saddle construction cannot be dismissed by assuming that "the prime function of a saddle is to give support to the artificial teeth." The requirements for the practice of dentistry have outgrown the stage of mechanics exclusively. The dental structures bear a fertile relativity of health or disease to the entire body.

II. CHART TWO

Objectives.—There are three general types of ridges, one being hard throughout, one soft throughout, and one consisting of hard and soft parts. Saddles, as they function on the ridges, serve a twofold purpose. For example, legs exist for body support, but they serve many purposes, as walking, dancing, standing, etc. Saddles for precision attachments are linked to position while supported by the alveolar ridge with intervening resilient tissue. At rest a saddle is seated in a relaxed position upon the ridge. In the action of mastication it functions upon the ridge.

[1] Charted for clinical demonstration.

Definitions.—"*Compression* is pathological of tissue and causes irritation. It is the total deformation, or the deformation per unit of length, of a substance, produced by a compressing force."—*Webster's Dictionary.* "*Displacement* is the act of displacing or state of being displaced. To displace is to change the place of or to take the place of."—*Webster's Dictionary.* "*Resilient* is synonymous with elastic, defined by buoyant."—*Webster's Dictionary.* "*The gums* are composed of dense fibrous tissue, closely connected to the periosteum of the alveolar processes and surrounding the necks of the teeth. They are covered by a smooth and vascular membrane, which is remarkable for its limited sensibility. Around the necks of the teeth the fibro-elastic portion presents numerous fine papillae; from here it is reflected into each alveolus, where it is continuous with the periosteal membrane lining that cavity."—*Gray's Anatomy.* "*Fibrous tissue* is the common connective tissue of the body and it is composed of yellow and white fibers."—*Dorland's American Pocket Medical Dictionary.*

III. CHART THREE

Relaxation is more difficult to explain than to experience. As it has been shown in golf, relaxation in action is at the basis of the science and art of the game.

If the tissue under a saddle is not relaxed while at rest, a compression of the underlying resilient tissue exists, with a consequent constant irritation of the soft and hard parts. It is generally thought that a chemical or mechanical trauma, either intermittent or constant, or of sufficient intensity, is a common cause of cancer. Dr. Tench has said: "The less stress on tissues, the more permanent the results."

Compression is not to be confused with displacement, such as exists in the relationship of muscles to the periphery of dentures and saddles, and in the displacement or proper placement of soft parts of a ridge nearer to the alveolar ridge under a saddle or denture.

IV. CHART FOUR

In visualizing the difference between compression and displacement, two apparently ambiguous but prognostically contradictory forces are involved. Irritation and stimulation both blanch the resilient

tissue under the saddle. The irritation results in inflammation and its train of pathology. The stimulation heightens the health quotient of the environment. Ethyl alcohol in its action presents a striking analogy. "Alcohol belongs, physiologically as well as chemically, to the group of aliphatic narcotics. In small doses it is a cerebral,

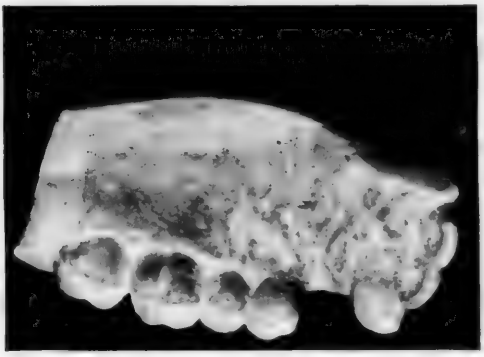

FIG. 1. THE CASE BEFORE TREATMENT

FIG. 2. THE ABUTMENT PREPARATIONS WITH THEIR RESPECTIVE INLAYS

respiratory, cardiac, and muscular stimulant; in large doses it is a depressant."—*United States Dispensary.*

Circulatory accommodations are pathological under irritation, physiological under stimulation.

It is decidedly unreasonable to attempt to produce sufficient compression of the ridge mucous-membrane to result in an actual alveolar-

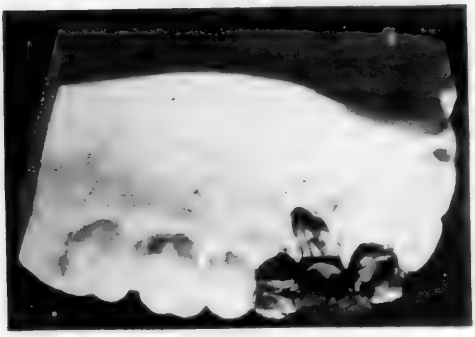

FIG. 3. THE LOCKS IN THEIR RESPECTIVE INLAYS AND WAXED TO THE SADDLE

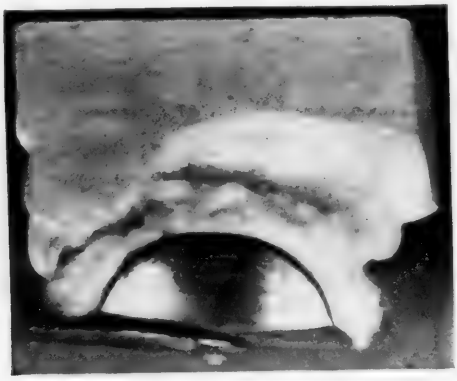

FIG. 4. A PLASTER IMPRESSION OF THE PREVIOUS STEP

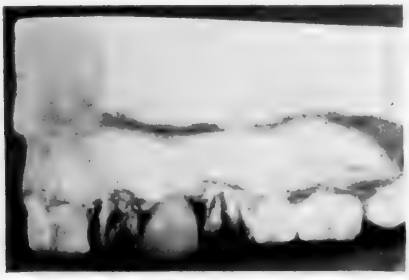

FIG. 5. THE FINISHED CASE

ridge seat for the saddle. This membrane is fibrous and buoyant, and in life has supporting not collapsible power. Therefore the resilient tissue must be relaxed while the saddle is at rest, and in function it will then be stimulated. If at rest the resilient tissue under a saddle is compressed, it will be irritated both at rest and in function.

It is therefore our aim to maintain a firm alveolar ridge and crest, and an uninflamed resilient ridge tissue and gingiva.

V. CHART FIVE

We shall now proceed to the physiological outline of a saddle. The saddle must leave free the gingival crest of the abutment teeth approximally, mesially, and distally, on the top of the ridge. Lingually the saddle must be as long as the lost roots. Facially the saddle must be long enough for the tooth it carries, rather than the saddle itself, to replace the lost process. Undercuts on the ridge must not be included, or the waxed saddle cannot be removed from the ridge without distortion.

It must be borne in mind that a healthy environment of the abutment roots is a necessary qualification for a firm gingival crest, an uninflamed gingiva, and for the success of the bridge. In the technique for a physiological saddle there is only one deviation from that outlined by Dr. H. E. S. Chayes in "Movable Removable Bridgework."

The usual plaster impression with the abutment inlays is taken and the model carefully made. The jackets of the attachments are soldered in the inlays in a parallel position to each other; the saddle with its mesial and distal thrusts is waxed and cast. Slots are made in the thrusts for the reception of the wings of the locks of the attachments. *The next step deviates from the present technique and requires the presence of the patient. The inlays containing the soldered jackets are placed in the mouth. The saddle is placed on the ridge. The locks are inserted in their respective inlays, with the wings of the attachments also in their respective slots. The fit of the saddle to the ridge is adjusted by waxing the wing to the thrust at its cervical end within the tooth part of the saddle. A plaster impression of the case, as now seated in the mouth, is taken. A second model is made, and the case finished as usual.*

885 West End Avenue.

BONE CHANGES INDUCED BY PERIODONTIC TREATMENT

Radiographic and Clinical Case Record

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Patient Miss G., Italian, thirty-two years of age, presented for treatment on November 15, 1926, with a severe gingivitis, complicated by marked mobility of teeth, and deep suppurative periodontal pockets. She had been advised to have all the teeth removed and replaced by dentures.

Clinical observations

Gingival tissues were decidedly congested and bloated, and patient complained of itching.

Suppuration was in evidence on slight pressure.

Traumatic occlusion and occlusal trauma were present.

Patient's general physical condition, as reported by a physician, was normal.

Periodontal pockets were observed around every tooth.

Mobility of teeth was more marked on the anterior teeth than on the posterior.

There was an odor of the breath characteristic of suppurative conditions. Saliva: slightly acid.

Radiographic observations

Marked alveolar absorption about all the teeth.

Bone condensation quite marked in the upper bicuspid and molar regions, right and left sides.

Extreme loss of bone on the lower anterior teeth.

Treatment

Commenced treatment on November 15, 1926. This consisted of charting the case, taking impressions for study models, procuring radiograms, balancing the occlusion, instruction in tooth-brushing and check-up of same.

Later, instrumentation was instituted, using hoes, sickels, and curettes. During the course of the treatment sodium sulphide prep-

FIG. 1. CASE BEFORE TREATMENT; PROTRUSIVE POSITION
FIG. 2. CASE BEFORE TREATMENT; CENTRIC POSITION
FIG. 3. CASE AT COMPLETION

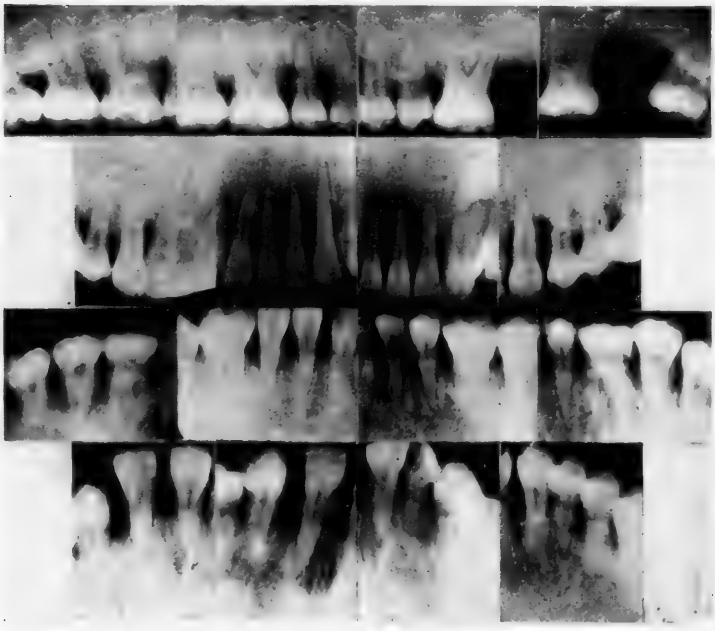

FIG. 4. RADIOGRAMS BEFORE TREATMENT

aration was used for aid in removing the epithelial tissue. She was dismissed on June 24, 1927, and told to report in three months.

When she returned, in October 1927 for re-examination, the mouth showed a return to health.

She was dismissed for six months, and further improvement was noted at that time.

When she returned again, on Dec. 8, 1928 for re-examination, the tissues still maintained their high state of health.

Definite results that had been observed

All suppuration had ceased.

Teeth had become firm.

Gingival tissue took on a healthy pink color, was free from congestion and patient made no further complaint of itching.

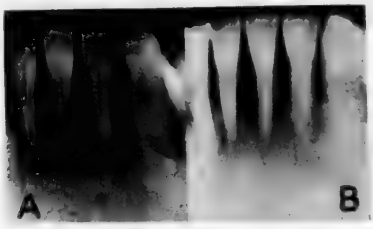

FIG. 5. BONE REGENERATION
A.—Before treatment. *B.*—Six months later

Periodontal pockets had closed.

Bone regeneration had taken place.

Abnormal bone condensation had disappeared between the upper left bicuspid and molar regions.

Patient was gratified by the fact that, for the first time in years, her mouth "felt good."

The balance of occlusion had a definite influence in restoring health in her mouth.

Further observations

A fact that is most interesting in this case is the gradual return to normal of the bone as observed in *fig. 6*—A, B, and C. This occurred after the balancing of occlusion had been completed. Another interesting fact is the filling in of healthy bone as observed in *fig. 6*, D, E, F, and G, and also in *fig. 5*.

A change in the contour of the gingival tissues in the completed case may also be noted (*fig. 3*). The health and tone of the tissues is very clearly demonstrated by the photograph.

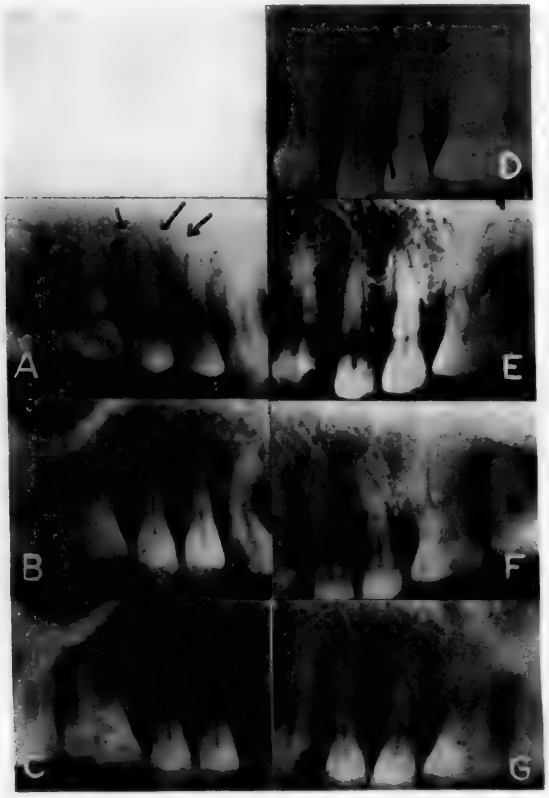

FIG. 6. RADIOGRAMS

Upper left: A.—Note bone condensation between bicuspids and first molar. *B.*—Note improvement within a year after relief of trauma. *C.*—Note healthy appearance of bone after two years.

Upper right: D, E.—Note bone condensation and rarefaction before treatment. The original film was lost; the illustrations show two photographic prints made from a lantern slide which had been preserved. The slight amounts of bone remaining between the bicuspids, and between the second bicuspid and first molar near the gingiva, are best shown in *D*. *F.*—Note filling-in of bone *one year* later, and reduction of condensation. (The right side could not be relieved properly, owing to the absence of the upper right lateral.) *G.*—Note further filling-in of bone *two years* later.

BIOMETRICS OF IDENTICAL TWINS FROM THE DENTAL VIEWPOINT[1]

SAMUEL GOLDBERG, M.S., L.D.S., D.D.S.

Chicago, Illinois

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I. PROLOGEMENA

Although the application of data on twins will contribute towards the solution of many dental problems, a field of study whose far reaching importance in every field of biological investigation has recently been stressed by several prominent directors of research in different countries, the author has confined his attention to the two issues which by common consent are the most urgent to the dental profession today; namely:

(1) A just vindication of the relative influence of the various forces that determine the shape of the dental arches, for this is the fundamental conception upon which the practice of orthodontia is based.

[1] Awarded the Morris L. Chaim and Benjamin J. Lord prizes, for 1928, by the First District Dental Society of the State of New York.

The dental examinations of the identical twins, studied in connection with the preparation of this paper, were made at Northwestern University Dental School and the results incorporated into a thesis on "A study of teeth and dental arches of identical twins." This thesis was presented in partial fulfillment of the requirements for the degree of Master of Science in Dentistry, which was conferred upon the author by Northwestern University on June 18, 1928. See abstract: *Journal of Dental Research*, 1929, ix, p. 273.

Since the completion of this work, the investigation has been extended not only in quantity but also in scope to fifty pairs of identical twins. This work was done in association with Professor Horatio Hackett Newman of the University of Chicago, who studied the same material from biological and psychological aspects for a book soon to be published.

(2) The determination of the presence of a peculiarity in tooth structure, of genetic origin, that predisposes to dental caries.

Apart from these main issues an attempt is being made to compare and reconcile various findings on the dentition with new findings on other characters recently investigated by Professor H. H. Newman.

II. HISTORICAL INTRODUCTION

The time-worn discussion of the relative influence of heredity and environment, on the various characters of man, might have defied solution for many more generations were it not for the unique opportunity afforded by nature in providing us with two identical individuals, both of whom have had a simultaneous start on the voyage of life, and who are generally assumed to carry the same hereditary mass. Arising from the dichotomy of the bilateral primordia of a single embryonic axis into two equal and coeval parts, each destined to become a new individual, neither the one nor the other can be construed as representing the original intention (1). The resemblance is both quantitative and qualitative in nature, the parallelism involving both chronological age and hereditary endowment.

The condition of any character at any given moment is the result of the action of three and only three factors; namely, *heredity* and *environment*, the resultant depending upon the *time* these interactions have proceeded. The impulsion toward growth and differentiation along definite paths, arising from a hereditary potential, is modified or enhanced by the influence of environment, within the limits set by death or total disfunction. Both heredity and environment vary, and successful development is not possible if either differs too much from "normal" conditions. Within these bounds set by death or disfunction, environment as well as hereditary structure may vary, so that the chance of meeting two individuals who are exactly alike is practically nil. Considering a single set of twenty-four chromosomes in man, each a bearer of one factor that is inherited independently of the other factors, the chance of getting two like combinations is one in 2×2^{24} or one in 32 million cases (2). The factors certainly are many more (even if partly coupled), and the chances of meeting two hereditarily like individuals, therefore, are exceedingly small. Besides there are the differences conditioned by

environment. As regards brothers and sisters, and fraternal twins, the chances of meeting individuals more nearly alike are much greater, but are still very remote.

A true conception of the relative influence of heredity, environment, and time is a matter of some moment to science, but in order to solve this, two factors in the equations must be known or equal quantities; and fortunately this latter requirement is fulfilled by uniovular twins. They furnish a unique opportunity of observing the development of hereditary characteristics, and comparing them with those resulting from the vagaries of this mortal existence. As aptly emphasized by Galton (3) in 1883: "Twins have a special claim upon our attention; it is that their history affords a means of distinguishing between the effects of tendencies received at birth and of those that were imposed by the special circumstances of their after-lives." Yet we find that, although from remote times many a fascinating piece of fiction has been built around identical twins, Galton's exhortation had until very recently received almost as little attention from the scientific world as the edible peas of Mendel attracted from 1866 to 1900 (although the "nature and nurture" phrase became common usage). It was not until 1920 that objective comparisons, mainly of a casual and fragmentary sort, began to creep into the literature.

If a character in an individual is such that it permits only a very small variational latitude under the influence of environment, it is said to be "hereditarily conditioned." Therefore, individuals in whom the hereditary factor conditioning the character is present always have the character, however environments may vary. If, however, a character manifests itself only under a certain environment, it is said to be "environmentally conditioned." Between these limits there may be characters partaking of these two extremes in any degree. The most measurably unalterable character known—that used for identification purposes—the finger prints and palm patterns is "hereditarily conditioned" and fixed at birth. Mental characteristics, on the other hand, judging from Prof. Newman's recent findings on identical twins reared apart, permit of very great alterations under environment. Where, between these most fixed and most labile characters, does the dental apparatus of man fall?

A true comprehension of the degree of similitude of identical twins

not only will permit a just vindication of the relative import of heredity and environment for every character in question, but also will elucidate the method of their origin. The characters that have received most attention are psychological and anatomical, although Crowden (4) reports a physiological study extending over the life time of a pair of identical twins.

During the earliest stages of growth and differentiation, epithelium is the dominant tissue, whose development precedes and controls that of other tissues (5). Hence, homologous epithelial structures are of particular interest to students of twin phenomena. Sano studied the brains of still-born monochorionic twins, and found them very much alike in structure (6). Siemens studied handedness (an epithelial character), and found nine opposite handed amongst forty-five pairs of identical twins (7). Prof. Newman, in a recent unpublished criticism, states that Siemens' criteria of handedness were unsatisfactory, being entirely subjective; and that judging from his own material, where the character in question was determined by actual tests, in which many avowedly right-handed subjects were in reality hereditarily left-handed but had changed through early training, the incidence of opposite handedness (partial and complete) is much greater. Opposite handedness is of significance in furnishing a clue to the incidence of inverse symmetry or "mirror-imaging," the recognition and frequency of which is of great importance in executing biometric work on identical twins. In the work done by Bachrach and Young (8)[2], no statement referring to the possible error introduced by disregarding this phenomenon is made; hence, their conclusions are to that extent unreliable. This is not surprising, in view of the total absence of any allusion in any literature to mirror-imaging in the jaws of identical twins. If the biological inferences with regard to other characters may be applied to the teeth, it can be stated that the inverse symmetry may be partial or complete, and the most cautious means of determining the extent of this inverse relation would be study casts of the dental arches. Moreover, Prof. Newman has found that inverse symmetry involves structures in proportion to their deep-seated or superficial character, the extent of involvement

[2] In quest of a hereditary factor in dental caries.

depending upon the degree of bilateral differentiation that has occurred around the primary axis before the dichotomy of the lateral halves. In conjoined twins this dichotomy occurs so late that a total separation is not effected; hence the frequency of inverse symmetry even of the viscera, which ordinarily would be the last involved, and which occurs rarely in separate twins. Commencing with the patterns on the index fingers, symmetry reversal involves other fingers, palms and soles, hair whorl, disposition of body-hair, "master" eye, etc., up the scale to handedness and viscera. Where in this series do the dental arches appear? It is assumed they fall somewhere between finger prints and handedness, and if the deep-seated or superficial character of the primordium of the future dental arch is nearer that of the palm pattern than it is to the handedness primordium, mirror-imaging in the denture would be of frequent occurrence in identical twins. A preliminary report will be made of four isolated evidences.

Other characters in identical twins that have received special attention are the friction-ridge patterns of the fingers, palms and soles (9), configuration of ears (10), naevus formation (11), dimples (12), eye and hair color, clasping of hands (13), and anthropological measurements on a host of characters, such as length of head, breadth of head, frontal minimum diameter, face breadth, bigonial diameter, face length, stature, sternal height, height of symphysis, height of acromium, height of tip of middle finger, length of arm, length of trunk, biacromial diameter, and bicristal diameter.

Reports on the pathology of identical twins are fairly common, but according to Murray (14), the subject merits greater attention than it has received as a means of determining the extent of an inborn error, or hereditary predisposition to disease, which will sooner or later indicate its presence by definite symptoms. Conversely, diseases that occur in both twins are most probably due to an inborn error rather than to external circumstances; and diseases that occur in one and not in the other twin are probably due to environmental causes. In this way each individual becomes a "control" from which to gauge the co-twin. As a concrete illustration of this method of reasoning, the Bauzek twins, Violet and Adeline (diagnosed by Prof. Newman as identical twins) will be cited. Violet has had nearly all the diseases of childhood; namely, scarlet fever, measles, chicken pox, etc. She presents

the typical hypoplastic teeth corresponding to the age of occurrence of the chicken pox. On her own evidence and that of her parents and of an elder sister, Adeline, except for occasional alimentary disturbances and "colds," has remained free from all her sister's diseases, and there was not the vaguest suspicion of an exanthematous fever in her history. Yet she too has hypoplastic teeth, of a type and distribution identical with those of Violet. The home conditions were investigated, and were found far below par, poverty precluding adequate nourishment. The children looked ill-nourished; in fact one of the younger children appeared rickety. As they were deprived most of the time even of milk in their diet, it is almost inconceivable how the twins had escaped rickets. In fact as the family had always been in poor circumstances, one may entertain a rickety diathesis as extremely probable. Now alimentary disturbances and rickets are possible causes of hypoplastic teeth. Or, beneath the chicken pox in Violet, another factor, an underlying metabolic diathesis, may be presumed, which in Adeline produced hypoplastic teeth without the clinical manifestation of the exanthematous disease. Further reports of such cases would definitely settle the question.

III. METHOD AND RESULTS

In the foregoing section it was stated that in a pair of identical twins each individual is derived from the primordium that would normally produce the right and left halves of a bilateral individual. If this bilateral fission occurred before the right and left halves of the primary axis had fully differentiated, and had become asymmetrical, we would expect the corresponding sides of the two individuals to differ less than the two halves of either individual. It would be preferable to study this hypothesis from some character that is hereditarily conditioned: were environment to have any appreciable influence, that character would not be a safe criterion to use. The friction-ridge patterns of the palms, soles, and fingers are admirably adapted to this purpose. But why is it that the difference between the corresponding sides of identical twins varies for every pair, yet always remains less than the bilateral difference? The answer to this question brings us to the problem of criteria used in the diagnosis of a pair of twins, which is sometimes shrouded with difficulty. Every

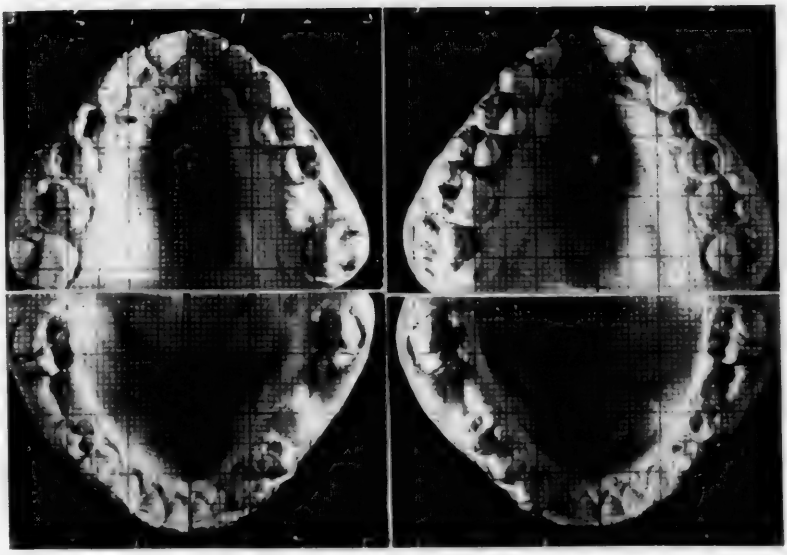

FIG. 1. PHOTOGRAPHS OF PLASTER MODELS: BAUZEK TWINS
Left.—Violet. *Right.*—Adeline

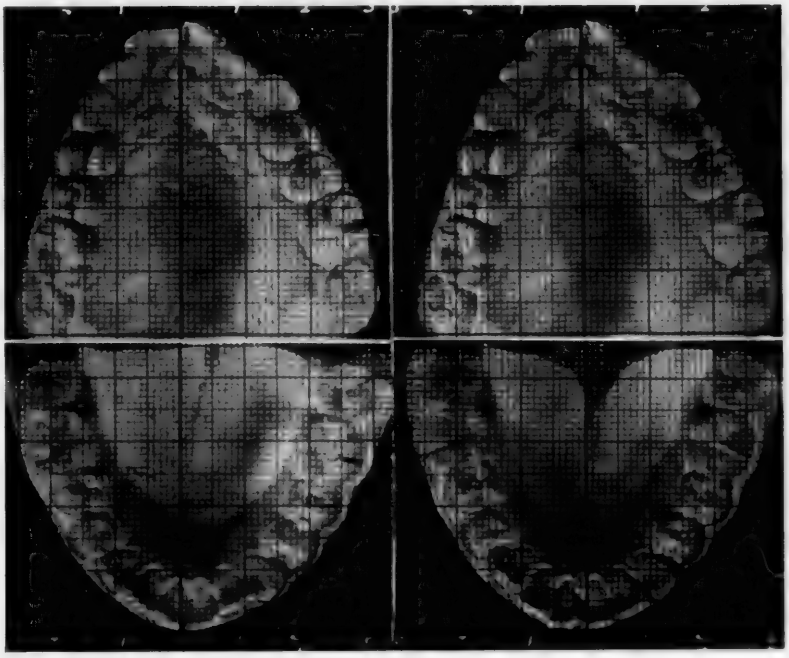

FIG. 2. PHOTOGRAPHS OF PLASTER MODELS: LARIMER TWINS
Left.—Evelyn. *Right.*—Katharine

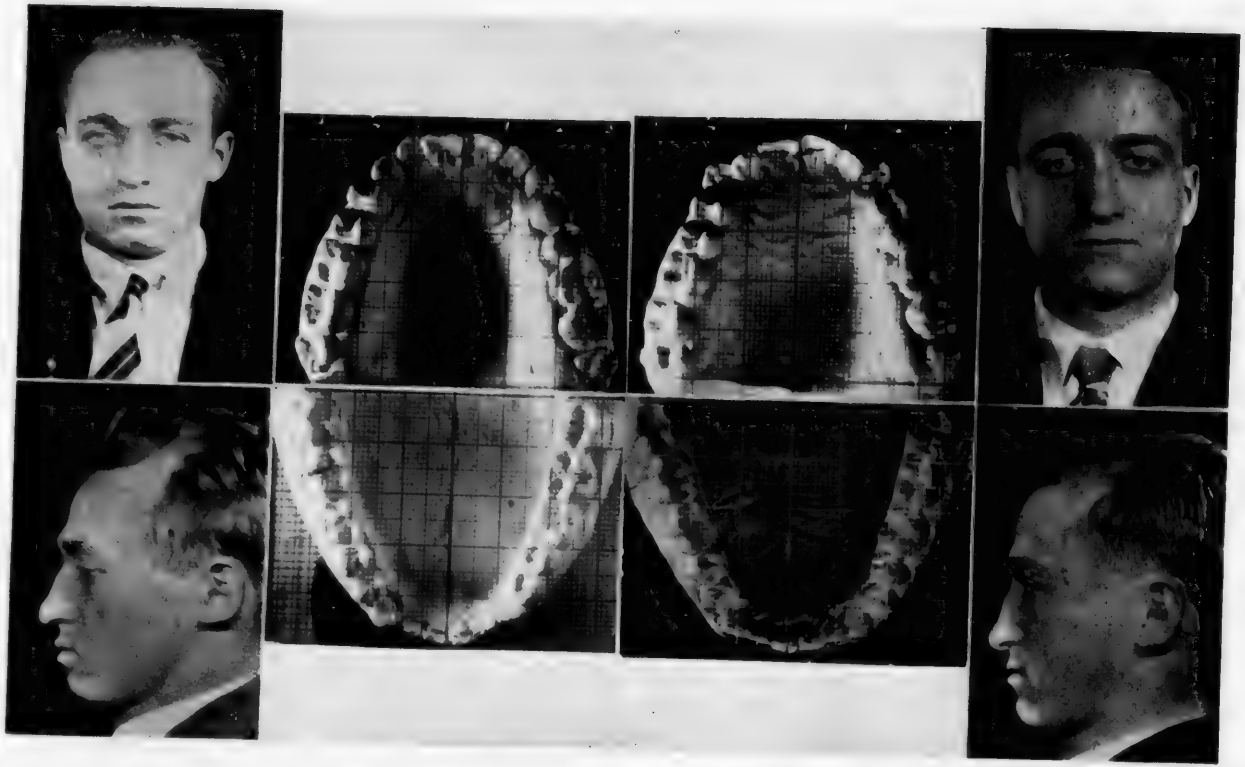

FIG. 3. PHOTOGRAPHS OF PLASTER MODELS: HUBATA TWINS
Left.—R. A. *Right.*—H. H.

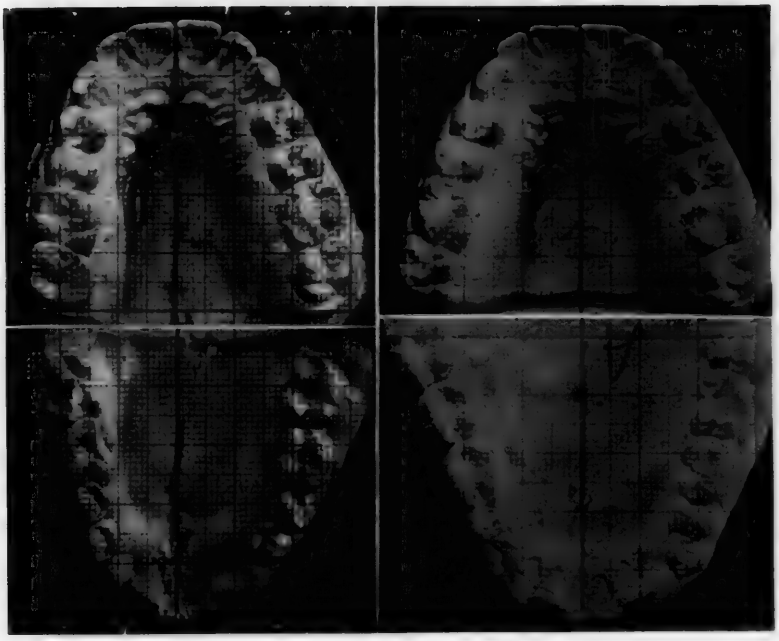

FIG. 4. PHOTOGRAPHS OF PLASTER MODELS: HOFFMAN TWINS
Left.—Raymond. *Right.*—Arthur

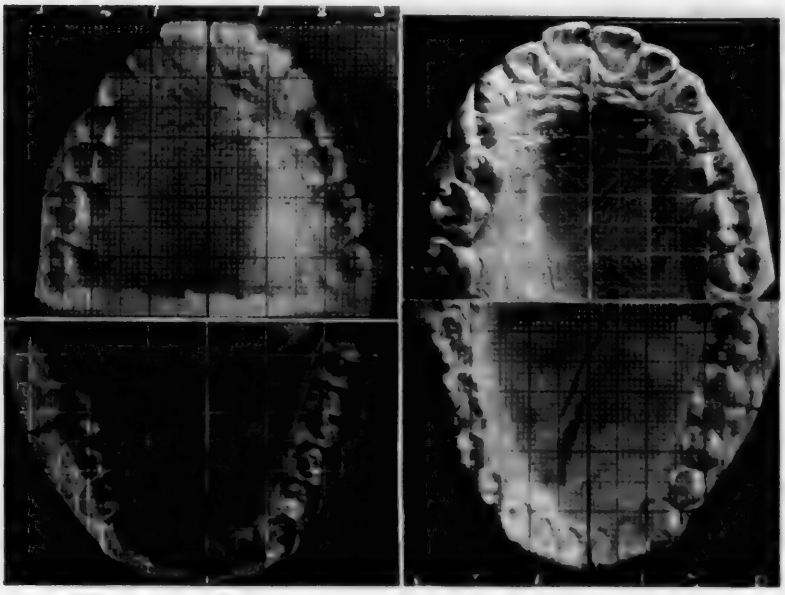

FIG. 5. PHOTOGRAPHS OF PLASTER MODELS: MERRILL TWINS
Left.—Garretson. *Right.*—Jackson

investigator in this field has recorded the difficulty and uncertainty attending the diagnosis of several of the least similar pairs of his group. According to the new and unpublished hypothesis propounded by Prof. H. H. Newman, a conclusion derived from a study of fifty pairs of identical twins, in which particular attention was given to the occurrence of inverse symmetry in a number of characters, the only

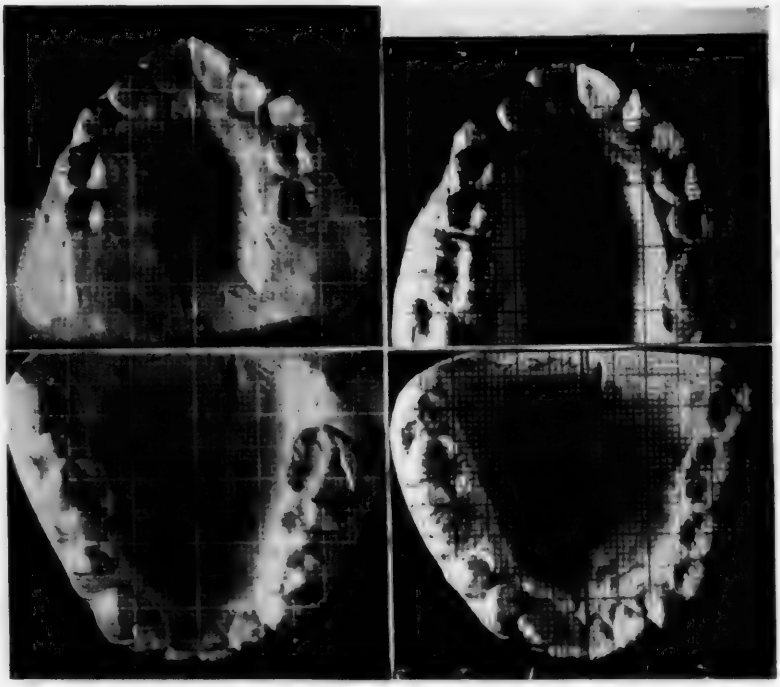

FIG. 6. PHOTOGRAPHS OF PLASTER MODELS: JUDD TWINS
Left.—Henry. *Right.*—Charles

inference possible is that the dichotomy of the bilateral primordia occurs at different stages of bilateral differentiation on both sides of the primary axis. Hence the difference between each pair of identical twins in any group varies, and it is unsafe to assume that they bear the same degree of resemblance to each other; in fact, they are soon to be classified into various grades in a forthcoming book by Prof. Newman,

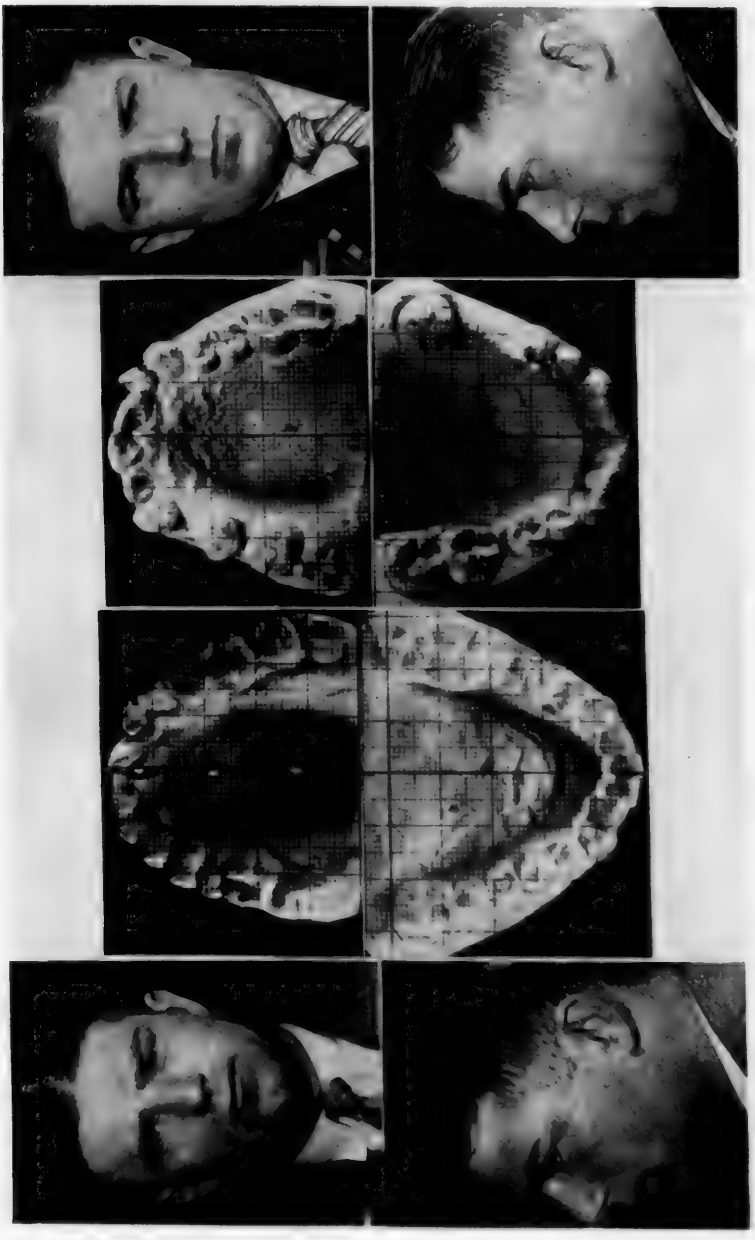

FIG. 7. PHOTOGRAPHS OF PLASTER MODELS: COURTS TWINS
Left.—W. A. *Right.*—J. J.

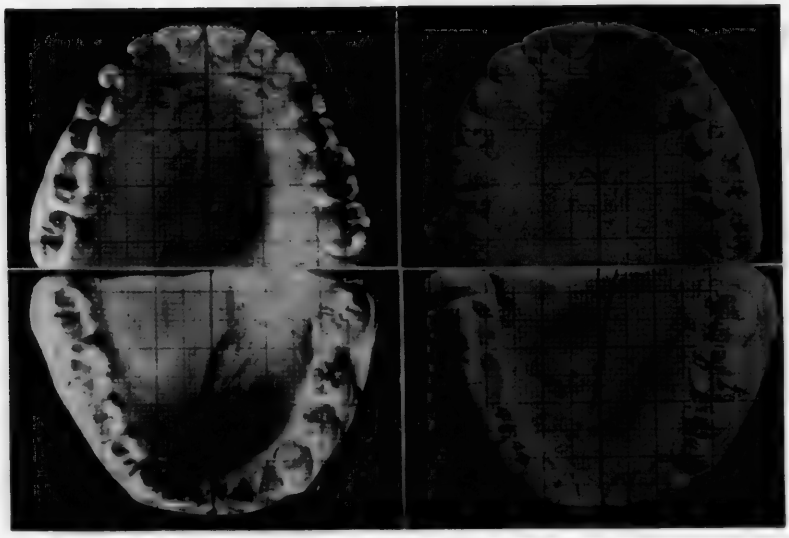

FIG. 8. PHOTOGRAPHS OF PLASTER MODELS: ALBIN TWINS
Left.—Arthur. *Right.*—Henry

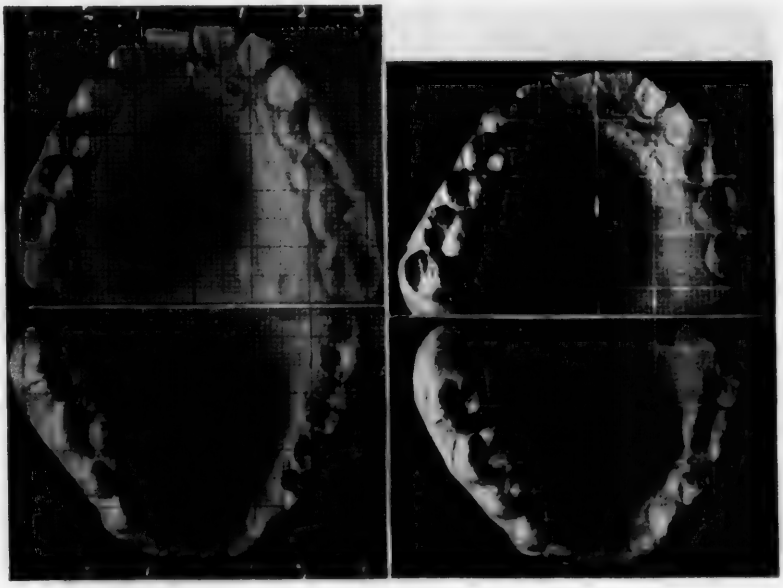

FIG. 9. PHOTOGRAPHS OF PLASTER MODELS: WEISS TWINS
Left.—Edward. *Right.*—Arthur

and it may be that a study of the dental arches will prove significant in the classification. The criteria in use at present are as follows:

(1) A single placenta or after-birth is incontrovertible, but rarely obtainable, evidence.

(2) Their appearance should give such an impression of great resemblance or "identity" to the visual senses that they have during

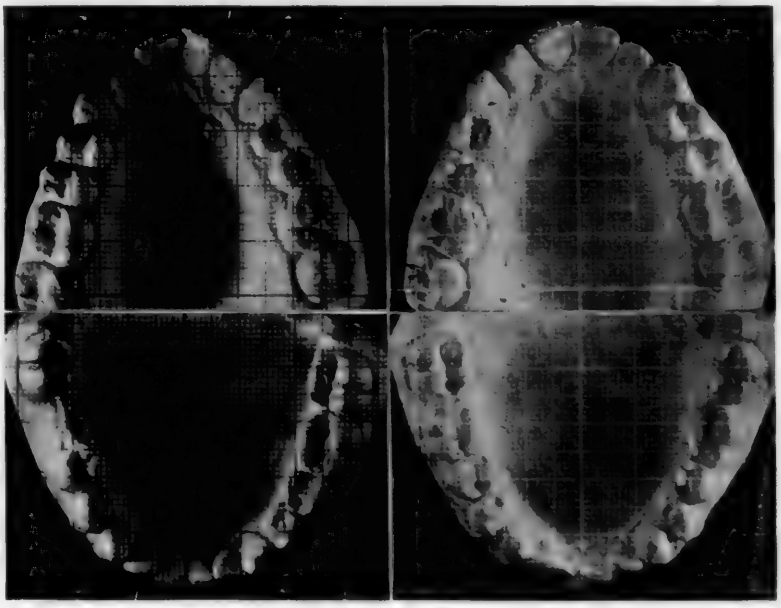

FIG. 10. PHOTOGRAPHS OF PLASTER MODELS: BUCHNER TWINS
Left.—H. O. *Right.*—H. J.

childhood been difficult to distinguish and have been confused even by neighbors, school fellows, and friends. Anent this subjective method, it may be said that the visual sense is more accurate in scrutinizing differences and similarities than any objective laboratory measurements can be.

(3) Anthropological measurements should not show too considerable differences.

FIG. 11. PHOTOGRAPHS OF PLASTER MODELS: NESBITT TWINS
Left.—Edward. *Right.*—Charles

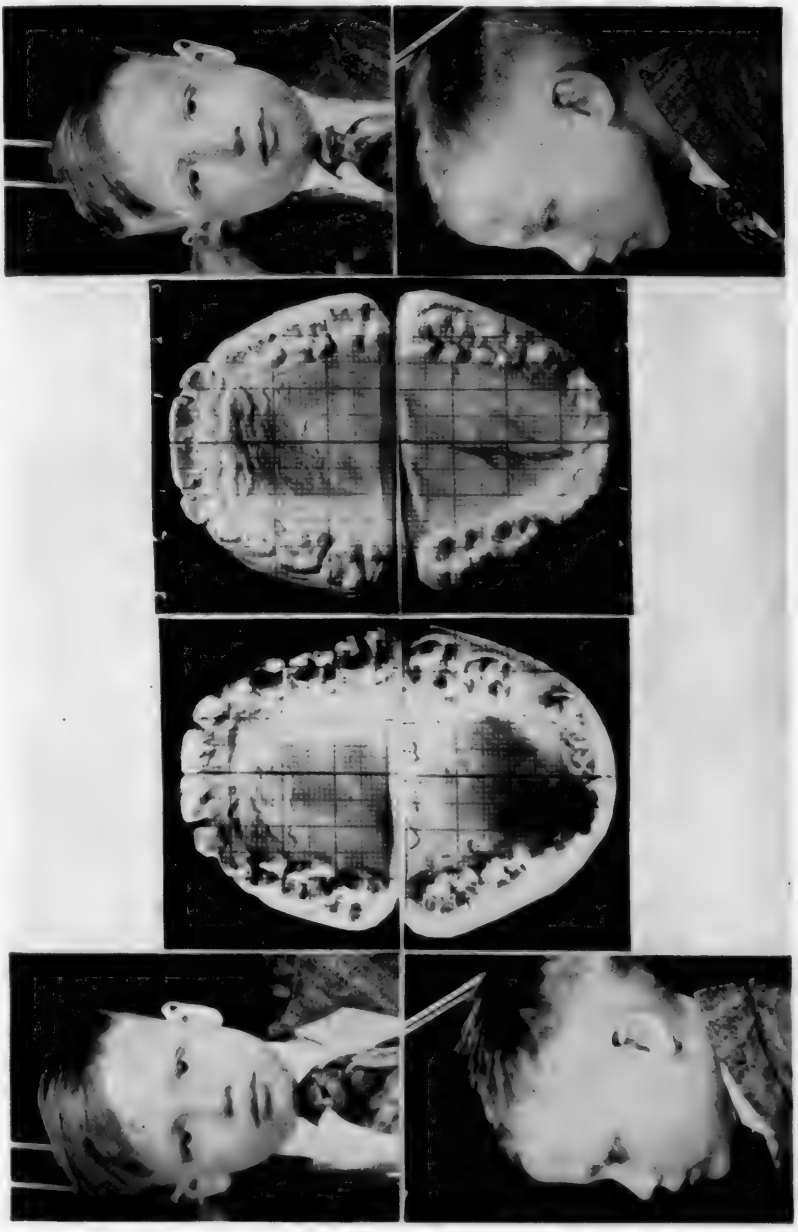

FIG. 12. PHOTOGRAPHS OF PLASTER MODELS: BEHRENS TWINS
Left.—Otto. *Right.*—George

FIG. 13. PHOTOGRAPHS OF PLASTER MODELS: ZWERKO TWINS
Left.—Casmir. *Right.*—Adam

(4) The configuration of the homologous[3] ears should not show greater dissimilarity than the left and right ear of each twin.

(5) The homologous finger prints, and palm and sole patterns, should not show greater differences than the right and left halves of each individual.

(6) The eye and hair color should be the same.

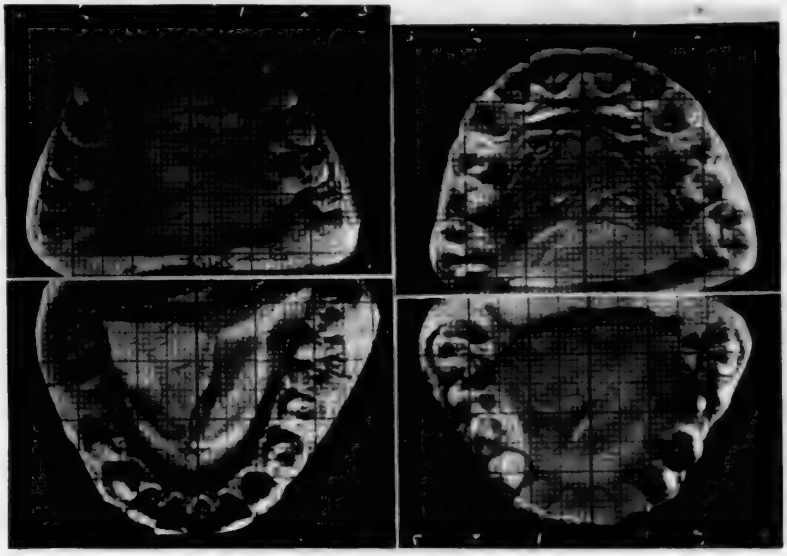

FIG. 14. PHOTOGRAPHS OF PLASTER MODELS: MCGRAW TWINS
Left.—Vivian. *Right.*—Lillian

Fifteen pairs of identical twins ranging in age from 3.5 to 24 years were studied. To fulfill the requirements of (1) and (2), full face and profile photographs showing the ears were taken whenever possible and appended. Palm and finger prints for every subject were obtained. From this evidence Prof. Newman diagnosed the fifteen pairs as identical twins.

Plaster impressions were taken and plaster casts made according to the methods in vogue among orthodontists. The median line of the

[3] By "homologous" is implied equivalence from the hereditary point of view.

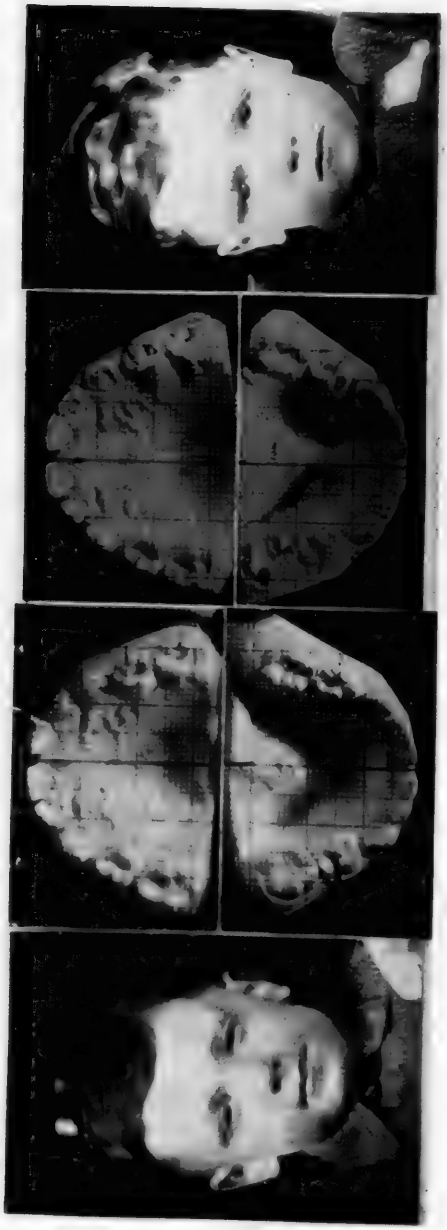

FIG. 15. PHOTOGRAPHS OF PLASTER MODELS: HEWETT TWINS
Left.—Craig. *Right.*—Paul

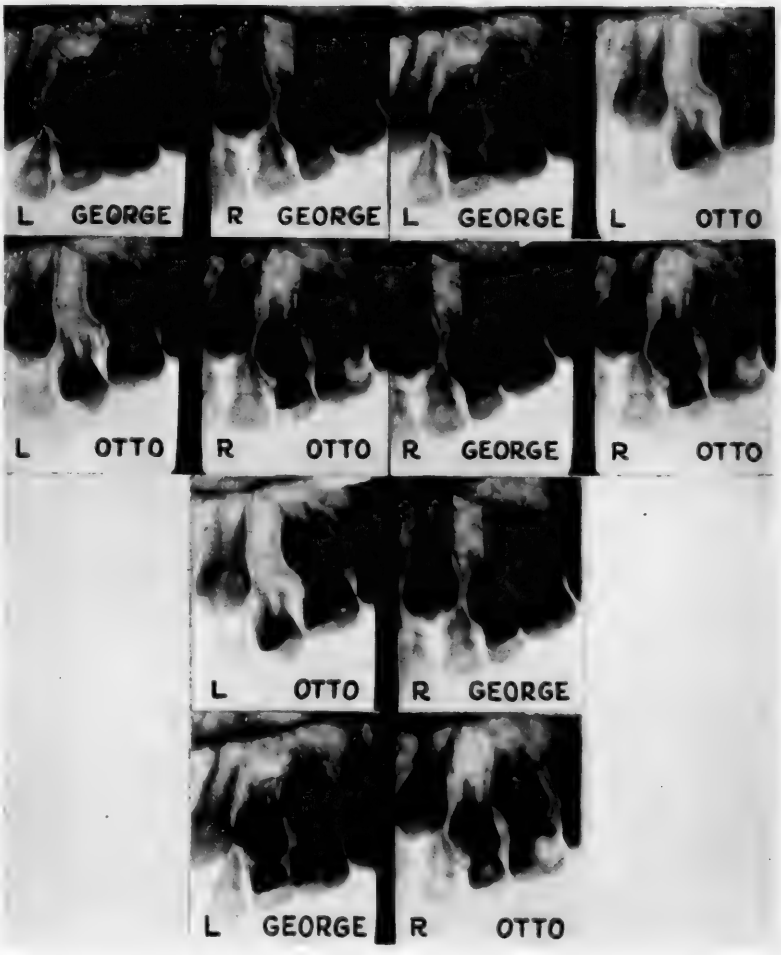

FIG. 16. X-RAY SHADOWGRAPHS OF THE TEETH OF THE BEHRENS TWINS (*fig. 12*)

In all these groupings, the right prints are reversed to facilitate comparisons.

The left of Otto is more like the right of George, than the left of Otto is like the right of Otto, or than the left of Otto is like the left of George, and vice versa. Also, *the left of George is more like the right of Otto*, than the right of George is like the left of George, or than the left of George is like the left of Otto, and vice versa.

TABLES 1–30

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (BLD) and the unilateral differences (ULD)

TABLES 1 AND 2: BAUZEK TWINS

TABLE 1.—MAXILLA

TOOTH	VIOLET				VIOLET		ADELINE		ADELINE				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	0	0	0.5	0	0	0	1	0	1	0	0	0	1	0	0.5
I²	7	2.5	7.5	3	0.5	0.5	0.5	1	7.5	4	7	3	0.5	1.5	0.5	0
C	13	7	13.5	8	0.5	0.5	1.5	1	12	10	13.5	9.5	1	1	0	1.5
B	17	15.5	18.5	15	1.5	1	1	2	17	17	18	15	0	1.5	0.5	0
B²	21	22	23	21	2	0.5	1.5	2.5	20.5	22.5	22	20	0.5	0.5	1	1
M	—	—	25	26	—	1	1.5	1.5	23.5	27	24.5	25.5	—	—	0.5	0.5
M²	26.5	35.5	27	35	1	—	2	3.5	26	37	28	33.5	0.5	—	1	1.5
	84.5	82.5	89.5	82.5	5.5	3.5	8	12.5	106.5	118.5	113	106.5	2.5	5.5	3.5	5

TABLE 2.—MANDIBLE

I	0	0	0	0	0	0	0	0.5	0	0	0	0.5	0	0	0	0.5
I²	5	1	5	1.5	0	0.5	0.5	0.5	5	1	4.5	1.5	0	0	0.5	0
C	10.5	5	11	6	0.5	1	0.5	0.5	11	5	10.5	4.5	0.5	0	0.5	1.5
B	15	9.5	15	11.5	0	2	0.5	1.5	15.5	9.5	15	11	0.5	0	0	0.5
B²	20	14	—	—	—	—	1.5	0.5	20	15	18.5	15.5	0	1	—	—
M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
M²	35.5	28	24.5	29	1	1	3	0.5	26	28.5	23	29	2.5	0.5	1.5	0
	66	57.5	55.5	48	1.5	4.5	6	4.0	77.5	59	71.5	62	3.5	1.5	2.5	2.5

TABLES 3 AND 4: LARIMER TWINS

TABLE 3.—MAXILLA

TOOTH	KATHARINE				KATHARINE		EVELYN		EVELYN				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0.5	0	−1	0.5	1.5	0.5	1	1.5	0	0	−1	1.5	0.5	0	0	1
I²	7	5.5	8	4.5	1	1	1	0	6.5	5.5	7.5	5.5	0.5	0	0.5	1
C	16	12	16.5	11.5	0.5	0.5	1.5	2.5	16.5	10.5	15	13	0.5	1.5	0.5	1.5
B	18.5	19	19.5	19	1	0	1.5	3	19.5	17.5	18	20.5	1	1.5	1.5	1.5
B²	22.5	26.5	22.5	26	0	0.5	4.5	3.5	23.5	24	19	27.5	1	2.5	3.5	1.5
M	25	32.5	23.5	33	1.5	0.5	5.5	3.5	27	13	21.5	33.5	2	2.5	2	0.5
M²	27	42	25.5	42.5	1.5	0.5	4.5	3.5	28.5	40	24	43.5	1.5	2	1.5	1
	116.5	137.5	114.5	137	7	3.5	19.5	17.5	121.5	127.5	104	145	7	10	9.5	8

TABLE 4.—MANDIBLE

I	1	0.5	−1	0	2	0.5	3	0.5	−1.5	0	1.5	0.5	2.5	0.5	2.5	0.5
I²	6.5	1.5	5	1.5	1.5	0	2.5	0.5	3.5	2	6	2.5	3	0.5	1	1
C	13	7.5	12.5	6.5	0.5	1	3	2	11.5	8	14.5	6	1.5	0.5	2	0.5
B	17.5	14	15.5	13	2	1	4.5	2.5	15	14	19.5	11.5	2.5	0	4	1.5
B²	19	21.5	19	19	0	2.5	3.5	3	17	21	21.5	18	2	0.5	2.5	1
M	21	27	21	26	0	1	4	2.5	20	27	24	24.5	1	0	3	1.5
M²	22.5	38	26.5	37	4	1	4	1.5	23	37	27	35.5	0.5	1	0.5	1.5
	100.5	110	98.5	103	10	7	24.5	12.5	88.5	109	114	98.5	13	3	15.5	7.5

TABLES 1–30—*Continued*

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (BLD) and the unilateral differences (ULD)

TABLES 5 AND 6: HUBATA TWINS

TABLE 5.—MAXILLA

TOOTH	H. H.				H. H.		R. A.		R. A.				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	0	0	1.5	0	1.5	0	1	0	0	0	1	0	0	0	0.5
I²	8.5	3.5	8	4.5	0.5	1	1.5	1.5	9.5	3	8	1.5	1	0.5	0	3
C	17	6	17	7	0	1	0	1.5	17	8	17	6.5	0	2	0	0.5
B	20.5	12.5	20	13.5	0.5	1	0.5	1.5	21.5	13	22	11.5	1	0.5	2	2
B²	24	18	22.5	20	1.5	2	1	2	24	19	25	17	0	1	2.5	3
M	27	23.5	25	25.5	2	2	0.5	2	26.5	26	27	24	0.5	2.5	2	1.5
M²	30	33	28.5	35	1.5	2	0.5	1	29	33.5	29.5	32.5	1	0.5	1	2.5
M³	33	43.5	29.5	45	3.5	1.5	1	1.5	32	44	31	42.5	1	0.5	1.5	2.5
	160	140	150.5	142	9.5	12	5	12	159.5	146.5	159.5	136.5	4.5	7.5	9	15.5

TABLE 6.—MANDIBLE

I	−2.5	0	2.5	1	4	1	2	0	−1	0	1	0	1.5	0	1.5	1
I²	3	1.5	7	1.5	3.5	0	0.5	1	5	1.5	5.5	2.5	2	0	1.5	1
C	11	4	14.5	3.5	4	0.5	0.5	1	12.5	4	13	5	1.5	0	1.5	1.5
B	15	9	19	9	3	0	0.5	1	17	9	16.5	10	2	0	2.5	1
B²	17.5	15.5	20.5	17	2.5	1.5	2	1	21	15.5	19	16.5	3.5	0	1.5	0.5
M	21	21.5	23.5	20.5	3	1	1.5	0	23.5	21	22	21	2.5	0.5	1.5	0.5
M²	24	30.5	27	31	0	0.5	2	0.5	26	30.5	25	31	2	0	2	0
M³	29	40.5	29	41	—	0.5	0.5	0.5	28.5	41	28	41.5	0.5	0.5	1	0.5
	118	122.5	143	124.5	25	5	9.5	0.5	132.5	122.5	130	127.5	15.5	1	14	6

TABLES 7 AND 8: HOFFMAN TWINS

TABLE 7.—MAXILLA

TOOTH	ARTHUR				ARTHUR		RAYMOND		RAYMOND				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	0	0	0.5	0	0.5	0	0.5	0	0	0	0.5	0	0	0	0
I²	8	2	9.5	1	1.5	1	0.5	0.5	8.5	1.5	9	1	0.5	0.5	0.5	0
C	16.5	9	16	8	0.5	1	0.5	0	16	8.5	15.5	8.5	0.5	0.5	0.5	0.5
B	21.5	17	21	15.5	0.5	1.5	1	0	21	16	20	16	0.5	1	1	0.5
B²	24	23	24.5	22	0.5	1	2.5	0	24	23	21.5	23	0	0	3	1
M	26	31	26	28	0	3	3	2.5	26	27.5	23	30	0	3.5	3	2
M²	29	39.5	30	33	1	6.5	3.5	1	28	38	24.5	39	1	1.5	5.5	6
	125	121.5	127	108	4	14.5	11	4.5	123.5	114.5	113.5	11.8	2.5	7	13.5	10

TABLE 8.—MANDIBLE

I	−1.5	0	1.5	1.5	3	1.5	4	1	−2	0	2	1	0.5	0	0.5	0.5
I²	3	3.5	6	4	3	0.5	3	1	3	3	6	4	0	0.5	0	0
C	11.5	6.5	13.5	5.5	2	0.5	2.5	1	11	6	13.5	5	0.5	0	0	0.5
B	15.5	11.5	18.5	11	3	0.5	2.5	1.5	15	13	17.5	11.5	0.5	1.5	1	0.5
B²	18	17.5	20	18	2	0.5	4.5	1	16	18.5	20.5	17.5	2	1	0.5	0.5
M	20	23	23.5	23.5	3.5	0.5	4	1	19	25	23	24	1	2	0.5	0.5
M²	23.5	33.5	25	35	1.5	1.5	4.5	0.5	21	36	25.5	35.5	2.5	2.5	0.5	0.5
M³	28.5	43.5	28.5	46	0	2.5	—	—	—	—	—	—	—	—	—	—
	118.5	139	136.5	144.5	18	8	25	7	83	101.5	108	98.5	7	75.	3	3

TABLES 1–30—*Continued*

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (BLD) and the unilateral differences (ULD)

TABLES 9 AND 10: MERRILL TWINS

TABLE 9.—MAXILLA

TOOTH	JACKSON				JACKSON		GARRETSON		GARRETSON				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	1	0	0	0	1	0	0.5	0	0.5	0	0	0	0.5	0	0
I^2	9.5	4	9	3.5	0.5	0.5	0	0	9	3.5	9	3.5	0.5	0.5	0	0
C	17.5	10	18	9.5	0.5	0.5	0.5	1	17	10	17.5	9	0.5	0	0.5	0.5
B	21	18	22	17	1	1	1	0.5	20	17.5	21	17	1	0.5	1	0
B^2	24	24.5	21	23	3	1.5	0.5	1	23	24	23.5	23	1	0.5	2.5	0
M	26	30	25.5	28	0.5	2	0.5	0.5	25	29.5	25.5	29	1	0.5	0	1
M^1	28	40.5	27.5	39	0.5	2	0.5	0	27	39.5	27.5	39.5	1	1	0	0.5
	126	128	123	120	6	8.5	3	3.5	121	124.5	124	121	5	3.5	4	2

TABLE 10.—MANDIBLE

I	1	0	−1	0	2	0	2	0	1	0	−1	0	0	0	0	0
I^2	6.5	1.5	5	1	1.5	0.5	1.5	0.5	6	1.5	4.5	1	0.5	0	0.5	0
C	14	6.5	13.5	5.5	0.5	1	1	0	14	5.5	13	5.5	0	1	0.5	0
B	18.5	13	17.5	12.5	1	0.5	2	1	18.5	13.5	16.5	12.5	0	0.5	1	0
B^2	22	19	20	19	2	0	1.5	0.5	21	19	19.5	18.5	1	0	0.5	0.5
M	23.5	24	23	25.5	0.5	1.5	1	0	23.5	25	22.5	25	0	1	0.5	0.5
M^1	25	35	25.5	36	0.5	1	1.5	0.5	26	35.5	24.5	35	1	0.5	1	1
	110.5	99	103.5	99.5	8	4.5	10.5	2.5	110	100	99.5	97.5	2.5	3	4	2

TABLES 11 AND 12: JUDD TWINS

TABLE 11.—MAXILLA

TOOTH	CHARLES				CHARLES		HENRY		HENRY				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	1	0	0	0	1	2	0.5	−1	0.5	1	0	1	0.5	1	0
I^2	9.5	4	8.5	4	1	0	2	0	7.5	5.5	9.5	5.5	2	1.5	1	1.5
C	15	11	16.5	8.5	1.5	2.5	1.5	1	15.5	10.5	17	11.5	0.5	0.5	0.5	3
B	18.5	18	18.5	15.5	0	2.5	2.5	0.5	17.5	19	20	19.5	1	1	1.5	4
B^2	20.5	24.5	20	22.5	0.5	2	0.5	2	19	25	20.5	27	1.5	0.5	0.5	4.5
	63.5	58.5	63.5	50.5	3	8	8.5	4	58.5	60.5	68	63.5	6	4	4.5	

TABLE 12.—MANDIBLE

I	0	1	0	0	0	1	0	0	0	0	0	0	0	1	0	0
I^2	6	2.5	5.5	2	0.5	0.5	0.5	0	6	1.5	5.5	1.5	0	1	0	0.5
C	14	8	12	5.5	2	2.5	0.5	1	12.5	6.5	13	5.5	1.5	1.5	1	0
B	17.5	14	17.5	11	0	3	0.5	2.5	17	14	16.5	11.5	0.5	0	1	0.5
B^2	—	—	20	18.5	—	—	—	—	—	—	18	20	—	—	2	1.5
M	23.5	27	21	26	2.5	1	0.5	2.5	18.5	21.5	19	27	5	5.5	2	1
M^1	—	—	24	37	—	—	—	—	23	33	—	—	—	—	—	—
	61	52.5	56	44.5	5	8	2	6	54	43.5	54	45.5	7	9	6	3.5

TABLES 1–30—*Continued*

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (BLD) and unilateral differences (ULD)

TABLES 13 AND 14: COURTS TWINS

TABLE 13.—MAXILLA

TOOTH	J. J.				J. J.		W. A.		W. A.				ULD			
	L		R		BLD		BLD		L		R		L		R	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	1	1	−1	0	2	1	2	1	−1	0	1	1	2	1	2	1
P^3	10.5	1.5	7.5	2.5	3.5	1	2	1.5	7.5	3	9.5	1.5	3.5	1.5	2.5	1
C	16.5	8.5	17	6	0.5	2.5	1.5	0	15.5	7.5	17	7.5	1	1	0	1.5
B	19	16	—	—	—	—	1	3	20	15	21	12	1	1	0	—
B^3	21.5	22	23.5	18	2	4	—	—	—	—	24	18	—	—	0.5	—
M	—	—	—	—	1	1	2	3.5	22	19.5	24	23	—	—	—	—
M^3	25	26.5	24	25.5	0.5	1	1	3.5	26	29.5	27	33	3	3	3	7.5
M^3	26	36.5	26.5	35.5	—	—	—	—	—	—	—	—	2	4	—	—
	100.5	96	97.5	87.5	9.5	10.5	9.5	12.5	90	74.5	98.5	78	12.5	11.5	8	11

TABLE 14.—MANDIBLE

I	2	2	−3	0	5	2	2	1	1	1	−1	0	1	1	2	0
P^3	6.5	3.5	2	3.5	4.5	0	1	2.5	5	4.5	4	2	1.5	1	2	1.5
C	14	8	9.5	6.5	4.5	0	3.5	1	8.5	8	12	7	5.5	0	2.5	0.5
B	17	14	14	11	3	3	2	0.5	17	14.5	15	14	0	0.5	1	3
B^3	20	21	17	17	3	4	1	2.5	20	20.5	19	18	0	0.5	2	
M	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
M^3	23	28.5	—	—	—	—	—	—	—	—	24.5	32	—	—	—	—
M^3	25	38.5	23	36.5	2	2	0.5	2	27	41	26.5	43	2	2.5	3.5	6.5
	84.5	87	62.5	74.5	22	11	10	9.5	78.5	89.5	75.5	84	10	5.5	13	12.5

TABLES 15 AND 16: ALBIN TWINS

TABLE 15.—MAXILLA

TOOTH	HENRY				HENRY		ARTHUR		ARTHUR				ULD			
	L		R		BLD		BLD		L		R		L		R	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	0	0	0	0	0	0	0.5	0	0	0	0.5	0	0	0	0.5
P^3	9	1	9	0.5	0	0.5	0.5	0.5	9.5	1	9	1.5	0.5	0	0	1
C	16.5	7.5	17.5	5.5	1	2	0.5	2	16.5	6	17	8	0	1.5	0.5	2.5
B	20.5	13	21.5	11.5	1	1.5	1	1	21	13	22	14	0.5	0	0.5	2.5
B^3	22.5	19	24	17	1.5	2	2	0.5	23	19.5	25	20	0.5	0.5	1	3
M	24.5	24	27	23	2.5	1	0.5	1	26	24	26.5	25	1.5	0	0.5	2
M^3	26	35.5	27.5	34.5	1.5	1	0	1	29	34	29	35	3	1.5	1.5	0.5
	119	100	126.5	92	7.5	8	4.5	6.5	125	97.5	128.5	104	6	3.5	4	12

TABLE 16.—MANDIBLE

I	0	0	0	0	4	0.5	0	0	−2	0	2	0.5	2	0	2	0.5
P^3	4.5	5	5	0.5	4.5	1	0.5	0	3.5	0	8	1	1	0.5	3	0.5
C	12	3	12.5	3.5	4.5	3	0.5	0.5	11.5	3	16	6	0.5	3	3.5	2.5
B	17	8	16.5	8.5	2.5	4.5	0.5	0.5	17.5	8.5	20	13	0.5	0.5	3.5	4.5
B^3	20	15	19.5	15.5	1	5.5	0.5	0.5	21	14.5	22	20	1	0.5	2.5	4.5
M	22.5	20.5	22	21.5	—	—	0.5	1	23	20.5	—	—	0.5	0	—	—
M^3	24	31	23	31.5	2.5	1	1	0.5	26.5	31.5	24	30.5	2.5	0.5	1	1
	100	82.5	98.5	81	19	15.5	3.5	3	78	57.5	92	75.5	8	5	15.5	13.5

TABLES 1-30—*Continued*

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (BLD) and the unilateral differences (ULD)

TABLES 17 AND 18: WEISS TWINS

TABLE 17.—MAXILLA

TOOTH	ARTHUR				ARTHUR		EDWARD		EDWARD				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	0.5	0	0	0	0.5	0	1	0.5	0	0.5	1	0.5	0.5	0.5	1
I²	9	2	8	2.5	1	0.5	1	1.5	10.5	1.5	9.5	3	1.5	0.5	1.5	0.5
C	17	8.5	16.5	7.5	0.5	1	0.5	0	18	9.5	17.5	9.5	1	1	1	2
B	20	17	21.5	14.5	1.5	2.5	2	1.5	22	17	24	18.5	2	0	2.5	2
B²	24	24	26.5	20.5	2.5	3.5	2	1.5	24.5	23	26.5	24.5	0.5	1	0	4
M	26	31	28.5	26	2.5	5	1.5	2.5	28	29	29.5	31.5	2	5	1	3.5
M²	—	—	34	35	—	—	—	—	30.5	39.5	—	—	—	—	—	—
	96	83	101	71	8	13	7	8	104.5	119.5	107.5	88	7.5	8	6.5	13

TABLE 18.—MANDIBLE

I	0	0.5	0	0	0	0.5	0	0.5	0	0.5	0	0	0	0	0	0
I²	5	1	6	0.5	1	0.5	1	1.5	5	1.5	6	0	0	0.5	0	0.5
C	12	5	12	6.5	0	1.5	1	1	12.5	6.5	11.5	5.5	0.5	1.5	0.5	1
B	17	10	17	11.5	0	1.5	0.5	4	17.5	14	17	10	0.5	4	0	1.5
B²	—	—	—	—	—	—	—	—	—	—	20.5	16	—	—	—	—
M	22	15.5	21	20.5	1	5	2	3.5	22	20	24	23.5	0	4.5	3	3
M²	26	24.5	24	29.5	2	5	0.5	3	26	30.5	26.5	33.5	0	6	2.5	4
	82	56.5	80	68.5	4	14	5	13.5	83	73	85	67.5	1	16.5	6	10

TABLES 19 AND 20: BUCHNER TWINS

TABLE 19.—MAXILLA

TOOTH	H. J.				H. J.		H. O.		H. O.				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	X	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	−1	0	1	0	2	0	0	1.5	0	0	0	1.5	1	0	1	1.5
I²	8.5	4	10.5	9	2	5	2	1.5	7.5	5.5	9.5	7	1	1.5	1	2
C	17.5	9.5	18.5	11.5	1	2	1.5	1	16	11.5	17.5	12.5	1.5	2	1	1
B	22.5	17	22	19	0.5	2	1.5	1.5	20	19	21.5	20.5	2.5	2	0.5	1.5
B²	25.5	23.5	24.5	26	1	2.5	0	2	24.5	24.5	24.5	26.5	1	1	0	0.5
M	28	30	27	33	1	3	1	2.5	27	31.5	26	34	1	1.5	1	1
M²	31	41.5	30.5	43	0.5	1.5	2	3.5	31	42	29	45.5	0	—	1.5	3.5
	132	125.5	134	141.5	8	16	8	13.5	126	134	128	147.5	8	8	6	11

TABLE 20.—MANDIBLE

I	−0.5	0	0	0.5	0.5	0.5	0	0	0	0	0	0	0.5	0	0	0.5
I²	4	3.5	5	3.5	1	0	0.5	1	5	2.5	5.5	1.5	1	1	0.5	2
C	12	6.5	13.5	6.5	1.5	0	0	2.5	13	8	13	4.5	1	1.5	0.5	2
B	17.5	12	19	10.5	1.5	1.5	1.5	5	17.5	14	19	9	0	2	0	1.5
B²	21.5	19	22	17	0.5	2	3	4.5	20.5	20.5	23.5	16	1	1.5	1.5	1
M	24	25.5	25.5	23	1.5	2.5	0	—	—	—	27	22	—	—	1.5	1
M²	26.5	37	29	35	2.5	2	3	4	25	38	28	34	1.5	1	1	1
	105	93.5	114	100	9	8.5	8	17	81	83	89	65	5	7	5	8

TABLES 1–30—*Continued*

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (BLD) and the unilateral differences (ULD)

TABLES 21 AND 22: NESBITT TWINS

TABLE 21.—MAXILLA

TOOTH	EDWARD				EDWARD		CHARLES		CHARLES				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	−1	0.5	1	0	2	0.5	0	0	0	0	0	0	1	0.5	1	0
P	8	3	10	3.5	2	0.5	2	1	7.5	4.5	9.5	3.5	0.5	1.5	0.5	0
C	15	11	14	10	1	1	0.5	1	15.5	12	15	11	0.5	1	1	1
B	19.5	16	11.5	16.5	0	0.5	0.5	0	20.5	19	20	19	1	3	0.5	2.5
P²	23	24	20.5	23	2.5	1	3.5	1	24	26	20.5	25	1	2	0	2
M	24	31	24	31	0	0	0	0.5	25.5	32.5	25.5	33.5	1.5	1.5	1.5	2
	88.5	85.5	81	84	7.5	3.5	6.5	3.5	93	94	90.5	92	5.5	9.5	4.5	7.5

TABLE 22.—MANDIBLE

I	−1	0	1	0	2	0	2	0	−1	0	1	0	0	0	0	0
I²	5	1.5	7	1.5	2	0	0.5	1.5	5.5	1.5	6	0	0.5	0	1	1.5
C	12	5.5	13.5	6	1.5	5	0.5	0	12.5	6.5	13	6.5	0.5	1	0.5	0.5
B	14	13	16	14	2	1	1.5	1	16	14	17.5	13	2	1	1.5	1
P²	17.5	20	—	—	—	—	1.5	2	20	20	18.5	18	2.5	0	—	—
M	20.5	28	22	28	1.5	0	1.5	7.5	21	20	22.5	27.5	0.5	8	0.5	0.5
	50.5	48	59.5	49.5	9	1.5	7.5	12	74	61	78.5	65	6	10	3.5	3.5

TABLES 23 AND 24: BEHRENS TWINS

TABLE 23.—MAXILLA

TOOTH	OTTO				OTTO		GEORGE		GEORGE				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	0.5	1	0	1	0.5	0	0.5	0	0	0	0.5	0	0.5	1	0.5
P	10	3	11	3	1	0	1	1	10	3	11	2	0	0	0	1
C	16.5	10	18	9.5	1.5	0.5	1.5	1	16.5	10	18	9	0	0	0	0.5
B	21	17	21	14.5	0	2.5	0.5	1	19.5	14	20	15	1.5	1	1	0.5
P²	22	23.5	23	22	1	1.5	0.5	1.5	22	22.5	22.5	21	0	1	0.5	1
M	25	32	26.5	30	1.5	2	1.5	2	24.5	31	26	29	0.5	1	0.5	1
	94.5	86	100.5	79	6	7	5	7	92.5	82.5	97.5	76.5	2	3.5	3	4.5

TABLE 24.—MANDIBLE

I	0	0	0	0.5	0	0.5	0	0.5	0	0	0	0.5	0	0	0	0
P	5.5	0.5	7	2	1.5	1.5	0	0	5.5	1	5.5	1	0	0.5	1.5	1
C	12.5	6	12	7	0.5	1	—	—	12.5	5.5	—	—	0	0.5	—	—
II	—	—	—	—	—	—	—	—	—	—	16	11	—	—	—	—
P²	18.5	16	19	17.5	0.5	1.5	0.5	2.5	18.5	15	18	17.5	0	1	1	0
M	24	26	22	30	2	4	1.5	3	22.5	24.5	21.5	27.5	1.5	1.5	0.5	2.5
	60.5	48.5	60	57	4.5	8.5	2	6	46.5	40.5	45	46.5	1.5	3.5	3	3.5

TABLES 1–30—*Continued*

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (ULD) and the unilateral differences (ULD)

TABLES 25 AND 26: ZWERKO TWINS

TABLE 25.—MAXILLA

TOOTH	ADAM				ADAM		CASMIR		CASMIR				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	1	0.5	−0.5	0	1.5	0.5	1	1	−0.5	1	0.5	0	1.5	0.5	1	0
I^2	9.5	2.5	8.5	0.5	1	2	1	4	8.5	2	9.5	−2	1	0.5	1	2.5
C	18	10	17.5	7	0.5	3	0.5	2	17	7	175	5	1	3	0	2
B	20.5	18	21.5	16	1	2	0	1	20	14	20	13	0.5	4	1.5	3
B^2	23	25.5	24	22	1	3.5	0.5	3	22.5	21	23	18	0.5	4.5	1	4
M	26	32.5	26.5	29	0.5	3.5	0	1	25	26	25	25	1	6.5	1.5	4
M^3	—	—	—	—	—	—	2	2.5	27	36.5	29	34	—	—	—	—
	98	70	97.5	74.5	5.5	14.5	5	14.5	119.5	107.5	124.5	93	5.5	19	6	15.5

TABLE 26.—MANDIBLE

I	0	0	0	0	5.5	0.5	0	0	3	0.5	−2.5	0	3	0.5	2.5	0
I^2	7	0.5	6.5	1.5	6.5	1.5	0.5	1	8.5	3	2	1.5	1.5	2.5	4.5	0
C	15	6	14	7	2.5	4	1	1	14.5	9	12	5	0.5	3	2	2
B	17.5	14	17	14	3	4.5	0.5	0	19	15.5	16	11	1.5	1.5	1	3
B^2	22	19.5	19	20.5	4.5	5.5	3	1	21.5	23	17	17.5	0.5	3.5	2	3
M	23.5	25.5	21	21.5	3.5	5.5	2.5	4.5	25	27	21.5	21.5	1.5	1.5	0.5	0
M^3	27.5	36	27	36	4.5	5.5	0.5	0	26.5	37.5	22.5	32	1	1.5	4.5	4
	112.5	91.5	16.5	100.5	30	27	8	7.5	118	115.5	90	88.5	9.5	14	17	12

TABLES 27 AND 28: McGRAW TWINS

TABLE 27.—MAXILLA

TOOTH	LILLIAN				LILLIAN		VIVIAN		VIVIAN				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	0	0.5	0	0	0	0.5	0	0	0	0	0	0	0	0.5	0	0
I^2	9	3.5	8.5	3	0.5	0.5	0.5	0.5	4	2.5	9	2	0.5	1	0.5	1
C	16.5	10	17	8.5	0.5	1.5	1.5	1	16	9.5	17.5	8.5	0.5	0.5	0.5	0
B	20	17	19	16	1	1	0	1.5	20.5	16	20.5	17.5	2.5	1	1.5	1.5
B^2	22	23.5	22	22	0	1.5	0	3	22.5	22	22.5	25	0.5	1.5	0.5	3
M	24.5	29.5	23	29.5	1.5	0	2.5	2.5	25.5	28	23	30.5	1	1.5	0	1
	92	64	89.5	79	3.5	5	4.5	8.5	88.5	78	92.5	83.5	5	6	3	6.5

TABLE 28.—MANDIBLE

I	2	1	−2	0	4	1	3.5	1	2	1	−1.5	0	0	0	0.5	0
I^2	7	3.5	4	2.5	3	1	3	1.5	7.5	2	4.5	0.5	0.5	1.5	0.5	2
C	13.5	10.5	11.5	7.5	2	3	2.5	1.5	15	9	12.5	7.5	1.5	1.5	1	0
B	6	17.5	16	13	0	4.5	1.5	3	19	15.5	17.5	12.5	3	2	1.5	0.5
B^2	—	—	19	21	—	—	1	2.5	20.5	22.5	21.5	20	—	—	2.5	1
M	20	29.5	22	27	2	2.5	0	1	22	28	22	27	2	1.5	—	—
	58.5	62	51.5	50	11	12	11.5	10.5	86	78	76.5	67.5	7	7	6	3.5

TABLES 1–30—*Continued*

The actual measurements of every tooth of each pair of twins, on each axis (X and Y), and the derivation therefrom of the bilateral differences (BLD) and the unilateral differences (ULD)

TABLES 29 AND 30: HEWETT TWINS

TABLE 29.—MAXILLA

TOOTH	PAUL				PAUL		CRAIG		CRAIG				*ULD*			
	L		*R*		*BLD*		*BLD*		*L*		*R*		*L*		*R*	
	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y	X	Y
I	1.5	0	1	0	0.5	0	0	0.5	1	0.5	1	0	0.5	0.5	0	0
I¹	9	1.5	8	2	1	0.5	1	1.5	7	3	8	1.5	2	1.5	0	0.5
C	16	7	14.5	8	1.5	1	0.5	2	14	10	14.5	8	2	3	0	0
B	19.5	15	17.5	15.5	2	0.5	1.5	2	17.5	17	19	15	2	2	1.5	0.5
B²	23.5	23	20.5	23	3	0	2.5	2	20.5	24.5	23	22.5	3	1.5	2.5	0.5
	69.5	46.5	61.5	48.5	8	2	5.5	8	60	55	65.5	47	9.5	8.5	4	1.5

TABLE 30.—MANDIBLE

I	1.5	0	1.5	0	0	0	0.5	0	1.5	0	1	0	0	0	0.5	0
I¹	5	1.5	5.5	0.5	0	1	0.5	0.5	5	0.5	4.5	1	0	1	1	0.5
C	12	5.5	12	4.5	0	1	1	1	12.5	4	11.5	6	0.5	1.5	0.5	1.5
B	15.5	12.5	16.5	11.5	1	1	2	1	16.5	11	14.5	12	1	1.5	2	0.5
B²	17.5	20.5	19	19	1.5	1.5	1.5	1.5	19.5	18	18	19.5	2	2.5	1	0.5
	51.5	40	54	35.5	2.5	4.5	5.5	5	35	33.5	49.5	38.5	3.5	6.5	5	3

palate being easily discernible, the model was adjusted in Simon's Symmetrograph, and the median line drawn. Without changing its position, the mandibular model was articulated with it, and the median line of the maxillary model projected onto it. Full size photographs of these models were made, onto the prints of which was thrown a cross-ruled millimeter scale to facilitate taking the biometric records. (*Figs. 1–15.*)

The measurements on the teeth were recorded by Cortesian coordinates, the median line of the palate representing the Y axis, the X axis lining with the centrals at right angles to this. The following abbreviations were adopted for convenience:

BLD = Bilateral difference (as between right and left sides).

ULD = Unilateral difference (as between corresponding sides).

I, I¹, C, B, B²; M, M¹, M² = Incisor, lateral, canine, first bicuspid, second bicuspid, first molar, second molar, third molar.

L and R = Left and right sides.

Tables 1 to *30* represent, side by side, the actual measurements of

TABLE 31

The bilateral differences for the corresponding jaws of each pair of twins arrayed against the unilateral differences between the corresponding sides

NUMBER OF TWIN	BLD				ULD			
					L		R	
	X	Y	X	Y	X	Y	X	Y
Mandible								
1	1.5	4.5	6	4	3.5	1.5	2.5	2.5
2	10	7	24.5	12.5	13.5	3	15.5	7.5
3	25	5	9.5	5	15.5	1	14	6
4	18	8	25	7	7	7.5	3	3
5	8	4.5	10.5	2.5	2.5	3	4	2
6	5	8	2	6	7	9	6	3.5
7	22	12.5	10	9.5	10	5.5	13	12.5
8	19	15.5	3.5	3	8	5	15.5	13.5
9	4	14	5	13.5	1	16.5	6	10
10	9	8.5	8	17	5	7	5	8
11	9	1.5	7.5	12	6	10	3.5	3.5
12	4.5	8.5	2	6	1.5	3.5	3	3.5
13	30	26.5	8	7.5	9	14	17	12
14	11	12	11.5	10.5	6.5	7	6	3.5
15	2.5	4.5	5.5	5	3.5	6.5	5	3
Maxilla								
1	5.5	3.5	8	12.5	2.5	5.5	3.5	5
2	7	3.5	19.5	17.5	7	10	9.5	8
3	9.5	12	5	12	4.5	7.5	9	15.5
4	4	14.5	11	4.5	2.5	7	13.5	10
5	6	8.5	3	3.5	5	3.5	4	2
6	3.0	8.0	8.5	4	6	4	4.5	13
7	9.5	10.5	9.5	12.5	12.5	11.5	8	11
8	7.5	8	4.5	6.5	6	3.5	4	12
9	8	13	7	8	7.5	8	6.5	13
10	7.5	20	8	13.5	8	8	6	11
11	7.5	3.5	6.5	3.5	5.5	9.5	4.5	7
12	6	7	5	7	2	3.5	3	4.5
13	5.5	14.5	5	14.5	5.5	19	6	15.5
14	11	12	11.5	10.5	6.5	7	6	3.5
15	8	2	5.5	8	9.5	8.5	4	1.5

TABLE 32

The total bilateral differences and the unilateral differences for each jaw in each twin

NUMBER OF TWIN	MANDIBLE				MAXILLA			
	BLD		*ULD*		*BLD*		*ULD*	
	X	Y	X	Y	X	Y	X	Y
1	7.5	8.5	6	4	13.5	16	6	10.5
2	34.5	19.5	29	10.5	26.5	21	16.5	18
3	34.5	10	29.5	7	14.5	24	13.5	23
4	43	15	10	10.5	15	19	16	17
5	18.5	7	6.5	.5	9	12	9	5.5
6	7	14	13	12.5	11.5	12	10.5	17
7	32	22	23	18	19	23	20.5	22.5
8	22.5	18.5	23.5	18.5	12	14.5	10	15.5
9	9	27.5	7	26.5	15	21	14	21
10	17	25.5	10	15	15.5	33.5	14	19
11	16.5	13.5	9.5	13.5	14	7	10	16.5
12	6.5	14.5	4.5	7	11	14	5	8
13	38	34	26	26	10.5	29	11.5	34.5
14	22.5	22.5	12.5	10.5	22.5	22.5	12.5	10.5
15	8	9.5	8.5	9.5	13.5	10	13.5	10

TABLE 33

The total bilateral differences and the unilateral differences for each pair of twins

NUMBER OF TWIN	MANDIBLE AND MAXILLA			
	BLD		*ULD*	
	X	Y	X	Y
1	21	24.5	12	14.5
2	61	40.5	45.5	28.5
3	49	34	43	30
4	58	34	26	27.5
5	27.5	19	15.5	10.5
6	18.5	26	23.5	29.5
7	51	45	43.5	40.5
8	34.5	33	33.5	34
9	24	48.5	21	47.5
10	32.5	59	24	34
11	30.5	20.5	19.5	30
12	17.5	28.5	9.5	15
13	48.5	63	37.5	60.5
14	45	45	25	21
15	16.5	19.5	22	19.5
	535	540	401	442.5

TABLE 34 (in seven parts)

The bilateral differences and the unilateral differences grouped, in relation to the type of teeth for mandible and maxilla, separately for each pair of twins

A. Incisor

NUMBER OF TWIN	MANDIBLE							MAXILLA						
	BLD			*ULD*			Ratio	*BLD*			*ULD*			Ratio
	X	x	x	X	x	x		X	x	x	X	x	x	
1	0	0	0	0	0	0		0	0	0	0	0	0	
2	5	2	3	5	2.5	2.5		2.5	1.5	1	5	5	0	
3	6	4	2	3	1.5	1.5		0	0	0	0	0	0	
4	7	3	4	1	0.5	0.5		0	0	0	0	0	0	
5	4	2	2	0	0	0		0	0	0	0	0	0	
6	0	0	0	0	0	0		2	0	2	2	1	1	
7	7	5	2	3	1	2		4	2	2	4	2	2	
8	4	4	0	4	2	2		0	0	0	0	0	0	
9	0	0	0	0	0	0		0	0	0	1	0.5	0.5	
10	0.5	0.5	0	0.5	0.5	0		2	2	0	2	1	1	
11	4	2	2	0	0	0		2	2	0	2	1	1	
12	0	0	0	0	0	0		1	1	0	1	0	1	
13	5.5	5.5	0	5.5	3	2.5		2.5	1.5	1	2.5	1.5	1	
14	7.5	4	3.5	0.5	0	0.5		0	0	0	0	0	0	
15	0.5	0	0.5	0.5	0	0.5		0.5	0.5	0	0.5	0.5	0	
	51			23			0.45	16.5			15.5			0.94
	y	y	Y	y	y	Y		y	y	Y	y	y	Y	
1	0	0.5	0.5	0	0.5	0.5		0	1	1	1	0.5	1.5	
2	0.5	0.5	1	0.5	0.5	1		0.5	1.5	2	0	1	1	
3	1	0	1	0	1	1		1.5	1	2.5	0	0.5	0.5	
4	1.5	1	2.5	0	0.5	0.5		0.5	0.5	1	0	0	0	
5	0	0	0	0	0	0		1	0.5	1.5	0.5	0	0.5	
6	1	0	1	1	0	1		1	0.5	1.5	0.5	0	0.5	
7	2	1	3	1	0	1		1	1	2	1	1	2	
8	0.5	0	0.5	0	0.5	0.5		0	0.5	0.5	0	0.5	0.5	
9	0.5	0.5	1	0	0	0		0.5	1	1.5	0.5	1	1.5	
10	0.5	0	0.5	0	0.5	0.5		0	1.5	1.5	0	1.5	1.5	
11	0	0	0	0	0	0		0.5	0	0.5	0.5	0	0.5	
12	0.5	0.5	1	0	0	0		0.5	0.5	1	0.5	0.5	1	
13	0.5	0	0.5	0.5	0	0.5		0.5	1	1.5	0.5	0	0.5	
14	1	1	2	0	0	0		0.5	0	0.5	0.5	0	0.5	
15	0	0	0	0	0	0		0	0.5	0.5	0.5	0	0.5	
			14.5			6.5	0.45			19			12.5	0.66

TABLE 34—*Continued*

B. Lateral incisor

NUMBER OF TWIN	MANDIBLE							MAXILLA						
	BLD			*ULD*			Ratio	*BLD*			*ULD*			Ratio
	X	z	x	X	z	x		X	z	x	X	z	x	
1	0.5	0	0.5	0.5	0	0.5		1	0.5	0.5	1	0.5	0.5	
2	4	1.5	2.5	4	3	1		2	1	1	1	0.5	0.5	
3	4	3.5	0.5	3.5	2	1.5		2	0.5	1.5	1	1	0	
4	6	3	3	0	0	0		2	1.5	0.5	1	0.5	0.5	
5	3	1.5	1.5	1	0.5	0.5		0.5	0.5	0	0.5	0.5	0	
6	1	0.5	0.5	0	0	0		3	1	2	3	2	1	
7	5	4.5	1	3.5	1.5	2		5.5	3.5	2	6	3.5	2.5	
8	5	4.5	0.5	4	1	3		0.5	0	0.5	0.5	0.5	0	
9	2	1	1	0	0	0		2	1	1	3	1.5	1.5	
10	1.5	1	0.5	1.5	1	0.5		3.5	1.5	2	2	1	1	
11	2.5	2	0.5	1.5	0.5	1		4	2	2	1	0.5	0.5	
12	1.5	1.5	0	1.5	0	1.5		2	1	1	0	0	0	
13	7	6.5	0.5	6	1.5	4.5		2	1	1	2	1	1	
14	6	3	3	1	0.5	0.5		1	0.5	0.5	1	0.5	0.5	
15	0.5	0	0.5	1	0	1		2	1	1	2	2	0	
	49.5			29			0.59	33			25			0.76
	y	y	Y	y	y	Y		y	y	Y	y	y	Y	
1	0.5	0.5	1	0	0	0		0.5	1	1.5	1.5	0	1.5	
2	0	0.5	0.5	0.5	1	1.5		1	0	1	0	1	1	
3	0	1	1	0	1	1		1	1.5	2.5	0.5	3	8	
4	0.5	1	1.5	0.5	0	0.5		1	0.5	1.5	0.5	0	0.5	
5	0.5	0.5	1	0	0	0		0.5	0	0.5	0.5	0	0.5	
6	0.5	0	0.5	1	0.5	1.5		0	0	0	1.5	1.5	3	
7	0	2.5	2.5	1	1.5	2.5		1	1.5	2.5	1.5	1	2.5	
8	1	0	1	0.5	0.5	1		0.5	0.5	1	0	1	1	
9	0.5	1.5	2	0.5	0.5	1		0.5	1.5	2	0.5	0.5	1	
10	0	1	1	1	2	3		5	1.5	6.5	1.5	2	3	
11	0	1.5	1.5	0	1.5	1.5		0.5	1	1.5	1.5	0	1.5	
12	1.5	0	1.5	0.5	1	1.5		0	1	1	0	1	1	
13	1.5	1	2.5	2.5	0	2.5		2	4	6	0.5	2.5	3	
14	1	1.5	2.5	1.5	2	3.5		0.5	0.5	1	1	1	2	
15	1	0.5	1.5	1	0.5	1.5		0.5	1.5	2	1.5	0	1.5	
			21.5			22.5	1.05			30.5			29.5	0.97

TABLE 34—*Continued*

C. Canine

NUMBER OF TWIN	MANDIBLE							MAXILLA						
	BLD			*ULD*			Ratio	*BLD*			*ULD*			Ratio
	X	x	x	X	x	x		X	x	x	X	x	x	
1	1	0.5	0.5	1	0.5	0.5		2	0.5	1.5	1	1	0	
2	3.5	0.5	3	3.5	1.5	2		2	0.5	1.5	1	0.5	0.5	
3	4.5	4	3	3	1.5	1.5		0	0	0	0	0	0	
4	4.5	2	2.5	0.5	0.5	0		1	0.5	0.5	1	0.5	0.5	
5	1.5	0.5	1	0.5	0	0.5		1	0.5	0.5	1	0.5	0.5	
6	2.5	2	0.5	2.5	1.5	1		3	1.5	1.5	1	0.5	0.5	
7	8	4.5	3.5	8	5.5	2.5		2	0.5	1.5	1	1	0	
8	5	4.5	0.5	4	0.5	3.5		1.5	1	0.5	0.5	0	0.5	
9	1	0	1	1	0.5	0.5		1	0.5	0.5	2	1	1	
10	1.5	1.5	0	1.5	1	0.5		2.5	1	1.5	2.5	1.5	1	
11	2	1.5	0.5	1	0.5	0.5		1.5	1	0.5	1.5	0.5	1	
12	0.5	0.5	1	—	0	—		3	1.5	1.5	0	0	0	
13	3.5	2.5	1	2.5	0.5	2		1	0.5	0.5	1	1	0	
14	4.5	2	2.5	2.5	1.5	1		2	0.5	1.5	1	0.5	0.5	
15	1	0	1	1	0.5	0.5		2	1.5	0.5	2	2	0	
	44.5			32.5			0.73	25.5			14.5			0.57
	y	y	Y	y	y	Y		y	y	Y	y	y	Y	
1	1	0.5	1.5	0	1.5	1.5		0.5	1	1.5	1	1.5	2.5	
2	1	2	3	0.5	0.5	1		0.5	2.5	3	1.5	1.5	3	
3	0.5	1	1.5	0	1.5	1.5		1	1.5	2.5	2	0.5	2.5	
4	0.5	1	1.5	0	0.5	0.5		1	0	1	0.5	0.5	1	
5	1	0	1	1	0	1		0.5	1	1.5	0	0	0	
6	2.5	1	3.5	1.5	0	1.5		2.5	1	3.5	0.5	3	3.5	
7	0	1	1	0	0.5	0.5		2.5	0	2.5	1	1.5	2.5	
8	3	0.5	3.5	3	2.5	5.5		2	2	4	1.5	2.5	4	
9	1.5	1	2.5	1.5	1	2.5		1	0	1	1	2	3	
10	0	2.5	2.5	1.5	2	3.5		0.5	1	1.5	2	1	3	
11	0.5	0	0.5	1	0.5	1.5		1	1	2	1	1	2	
12	1	—	1	0.5	—	0.5		0.5	1	1.5	0	0.5	0.5	
13	4	1	5	3	2	5		3	2	5	3	2	5	
14	3	1.5	4.5	1.5	0	1.5		1.5	1	2.5	0.5	0	0.5	
15	1	2	3	1.5	1.5	3		1	2	3	3	0	0	
			35			25.5	0.73			36			36	1.00

TABLE 34—*Continued*

D. First bicuspid

NUMBER OF TWIN	MANDIBLE							MAXILLA						
	BLD			*ULD*			Ratio	*BLD*			*ULD*			Ratio
	X	x	x	X	x	x		X	x	x	X	x	x	
1	0.5	0	0.5	0.5	0.5	0		2.5	1.5	1	0.5	0	0.5	
2	6.5	2	4.5	6.5	2.5	4		2.5	1	1.5	2.5	1	1.5	
3	3.5	3	0.5	4.5	2	4.5		1	0.5	0.5	3	1	2	
4	5.5	3	2.5	1.5	0.5	1		1.5	0.5	1	1.5	0.5	1	
5	3	1	2	1	0	1		2	1	1	2	1	1	
6	0.5	0	0.5	1.5	0.5	1		2.5	0	2.5	2.5	1	1.5	
7	5	3	2	1	0	1		1	—	1	1	1	0	
8	3	2.5	0.5	4	0.5	3.5		2	1	1	1	0.5	0.5	
9	0.5	0	0.5	0.5	0.5	0		3.5	1.5	2	4.5	2	2.5	
10	3	1.5	1.5	0	0	0		2	0.5	1.5	3	2.5	0.5	
11	3.5	2	1.5	3.5	2	1.5		0.5	0	0.5	1.5	1	0.5	
12	—	—	—	—	—	—		0.5	0	0.5	2.5	1.5	1	
13	3.5	3	0.5	2.5	1.5	1		1	1	0	2	0.5	1.5	
14	1.5	0	1.5	4.5	3	1.5		1	1	0	4	2.5	1.5	
15	3	1	2	3	1	2		3.5	2	1.5	3.5	2	1.5	
	42.5			35.5			0.80	25			35			1.40
	y	y	Y	y	y	Y		y	y	Y	y	y	Y	
1	2	1.5	3.5	0	0.5	0.5		1	2	3	1.5	0	1.5	
2	1	2.5	3.5	0	1.5	1.5		0	3	3	1.5	1.5	3	
3	0	1	1	0	1	1		1	1.5	2.5	0.5	2	2.5	
4	0.5	1.5	2	1.5	0.5	2		1.5	0	1.5	1	0.5	1.5	
5	5	1	6	0.5	0	0.5		1	0.5	1.5	0.5	0	0.5	
6	3	2.5	5.5	0	0.5	0.5		2.5	0.5	3	1	4	5	
7	3	0.5	3.5	0.5	3	3.5		—	3	3	1	—	1	
8	4.5	0.5	5	0.5	4.5	5		1.5	1	2.5	0	2.5	2.5	
9	1.5	4	5.5	4	1.5	5.5		2.5	1.5	4	0	2	2	
10	1.5	5	6.5	2	1.5	3.5		5.5	1.5	7	2	1.5	3.5	
11	1	1	2	1	1	2		0.5	0	0.5	3	2.5	5.5	
12	—	—	—	—	—	—		2.5	1	3.5	1	0.5	1.5	
13	4.5	0	4.5	1.5	3	4.5		2	1	3	4	3	7	
14	4.5	3	7.5	2	0.5	2.5		1	1.5	2.5	1	1.5	2.5	
15	1	1	2	1.5	0.5	2		0.5	2	2.5	2	0.5	2.5	
			58			34.5	0.60			43			41.5	0.96

TABLE 34—*Continued*

E. Second biscupid

NUMBER OF TWIN	MANDIBLE							MAXILLA						
	BLD			*ULD*			Ratio	*BLD*			*ULD*			Ratio
	X	x	x	X	x	x		X	x	x	X	x	x	
1	1.5	0	1.5	0	0	0		3.5	2	1.5	1.5	0.5	1	
2	3.5	0	3.5	4.5	2	2.5		4.5	0	4.5	4.5	1	3.5	
3	4.5	2.5	2	5	3.5	1.5		2.5	1.5	1	2.5	0	2.5	
4	6.5	2	4.5	2.5	2	0.5		3	0.5	2.5	3	0	3	
5	3.5	2	1.5	1.5	1	0.5		3.5	3	0.5	2	1	1	
6	—	—	—	—	—	—		1	0.5	0.5	2	1.5	0.5	
7	4	3	1	2	0	2		2	2	—	0.5	—	0.5	
8	1.5	1	0.5	3.5	1	2.5		3.5	1.5	2	1.5	0.5	1	
9	—	—	—	—	—	—		4.5	2.5	2	0.5	0.5	0	
10	3.5	0.5	3	2.5	1	1.5		1	1	0	1	1	0	
11	1.5	1	1.5	2.5	2.5	—		6	2.5	3.5	1	1	2	
12	1	0.5	0.5	1	0	1		1.5	1	0.5	0.5	0	0.5	
13	7.5	4.5	3	2.5	0.5	2		1.5	1	0.5	1.5	0.5	1	
14	1	—	—	2.5	—	2.5		0	0	0	1	0.5	0.5	
15	3	1.5	1.5	3	2	1		5.5	3	2.5	5.5	3	2.5	
	42.5			33			0.78	43.5			28.5			0.66
	y	y	Y	y	y	Y		y	y	Y	y	y	Y	
1	0	0.5	0.5	0	1	1		0.5	2.5	3	0.5	1	1.5	
2	2.5	3	5.5	0.5	1	1.5		0.5	3.5	4	2.5	1.5	4	
3	1.5	1	2.5	0	0.5	0.5		2	2	4	1	3	4	
4	0.5	1	1.5	1	0.5	1.5		1	0	1	0	1	1	
5	0	0.5	0.5	0	0.5	0.5		1.5	1	2.5	0.5	0	0.5	
6	—	—	—	—	—	—		2	2	4	0.5	4.5	5	
7	4	2.5	6.5	0.5	1	1.5		4	—	4	—	—	—	
8	5.5	0.5	6	0.5	4.5	5		2	0.5	2.5	0.5	3	3.5	
9	—	—	—	—	—	—		3.5	1.5	5	1	4	5	
10	2	4.5	6.5	1.5	1.5	2		2	2	4	1	0.5	1.5	
11	—	2	2	0	—	0		1	1	2	2	2	4	
12	1.5	2.5	4	1	0	1		1.5	1.5	3	1	1	2	
13	5.5	1	6.5	3.5	3	6.5		3.5	3	6.5	4.5	4	8.5	
14	—	2.5	2.5	—	1	1		1.5	3	4.5	1.5	3	4.5	
15	1.5	1.5	3	2.5	0.5	3		0	2	2	1.5	0.5	2	
			47.5			22	0.46			50			47	0.94

TABLE 34—*Continued*

F. First molar

NUMBER OF TWIN	MANDIBLE							MAXILLA						
	BLD			*ULD*			Ratio	*BLD*			*ULD*			Ratio
	X	x	x	X	x	x		X	x	x	X	x	x	
1	—	—	—	—	—	—		1.5	—	1.5	0.5	—	0.5	
2	4	0	4	4	1	3		7	1.5	5.5	4	2	2	
3	4.5	3	1.5	4	2.5	1.5		2.5	2	0.5	2.5	0.5	2	
4	7.5	3.5	4	1.5	1	0.5		3	0	3	3	0	3	
5	1.5	0.5	1	0.5	0	0.5		1	0.5	0.5	1	1	0	
6	3	2.5	0.5	7	5	2		—	—	—	—	—	—	
7	—	—	—	—	—	—		3	1	2	—	—	—	
8	0.5	—	0.5	0.5	0.5	—		3	2.5	0.5	2	1.5	0.5	
9	3	1	2	3	0	3		4	2.5	1.5	3	2	1	
10	1.5	1.5	0	1.5	—	1.5		2	1	1	2	1	1	
11	3	1.5	1.5	1	0.5	0.5		0	0	0	3	1.5	1.5	
12	3.5	2	1.5	2	1.5	0.5		3	1.5	1.5	1	0.5	0.5	
13	6	3.5	2.5	2	1.5	0.5		0.5	0.5	0	2.5	1	1.5	
14	2	2	0	2	2	—		4	1.5	2.5	1	1	0	
15	—	—	—	—	—	—		—	—	—	—	—	—	
	40			29			0.73	34.5			25.5			0.74
	y	y	Y	y	y	Y		y	y	Y	y	y	Y	
1	—	—	—	—	—	—		1	1.5	2.5	—	0.5	0.5	
2	1	2.5	3.5	0	1.5	1.5		0.5	3.5	4	2.5	0.5	3	
3	1	0	1	0.5	0.5	1		2	2	4	2.5	1.5	3.5	
4	0.5	1	1.5	2	0.5	2.5		3	2.5	5.5	3.5	2	5.5	
5	1.5	0	1.5	1	0.5	1.5		2	0.5	2.5	0.5	1	1.5	
6	1	2.5	3.5	5.5	1	6.5		—	—	—	—	—	—	
7	—	—	—	—	—	—		1	3.5	4.5	—	—	—	
8	—	1	1	0	1	0		1	1	2	0	2	2	
9	0.5	3.5	4	4.5	3	7.5		5	2.5	7.5	5	3.5	8.5	
10	2.5	—	2.5	—	1	1		2.5	2.5	5	1.5	1	2.5	
11	0	7.5	7.5	8	0.5	8.5		0	0.5	0.5	1.5	2	3.5	
12	4	3	7	1.5	2.5	4		2	2	4	1	1	2	
13	5.5	4.5	10	1.5	0	1.5		3.5	1	4.5	6.5	4	10.5	
14	2.5	1	3.5	1.5	—	1.5		0	2.5	2.5	1.5	1	2.5	
15	—	—	—	—	—	—		—	—	—	—	—	—	
			46.5			37	0.80			49			45.5	0.93

TABLE 34—*Concluded*

G. Second molar

NUMBER OF TWIN	MANDIBLE							MAXILLA						
	BLD			*ULD*			Ratio	*BLD*			*ULD*			Ratio
	X	x	x	X	x	x		X	x	x	X	x	x	
1	4	1	3	3	2.5	1.5		3	1	2	1.5	0.5	1	
2	8	4	4	1	0.5	0.5		6	1.5	4.5	3	1.5	1.5	
3	2	0	2	4	2	2		2	1.5	0.5	2	1	1	
4	6	1.5	4.5	3	2.5	0.5		4.5	1	3.5	6.5	1	5.5	
5	2	0.5	1.5	2	1	1		1	0.5	0.5	1	1	0	
6	—	—	—	—	—	—		—	—	—	—	—	—	
7	—	—	—	—	—	—		1.5	0.5	1	6	3	3	
8	3.5	2.5	1	3.5	2.5	1		1.5	1.5	0	4.5	3	1.5	
9	2.5	2	0.5	2.5	0	2.5		—	—	—	—	—	—	
10	5.5	2.5	3	2.5	1.5	1		2.5	0.5	2	1.5	0	1.5	
11	—	—	—	—	—	—		—	—	—	—	—	—	
12	—	—	—	—	—	—		—	—	—	—	—	—	
13	5	4.5	0.5	5.5	1	4.5		2	—	2	—	—	—	
14	—	—	—	—	—	—		—	—	—	—	—	—	
15	—	—	—	—	—	—		—	—	—	—	—	—	
	38.5			27			0.77	24			26			1.08
	y	y	Y	y	y	Y		y	y	Y	y	y	Y	
1	1	0.5	1.5	5	0	5		—	3.5	3.5	—	1.5	1.5	
2	1	1.5	2.5	1	1.5	2.5		0.5	3.5	4	2	1	3	
3	0.5	0.5	1	0	0	0		2	1	3	0.5	2.5	5	
4	1.5	0.5	2	2.5	0.5	3		6.5	1	7.5	1.5	6	7.5	
5	1	0.5	1.5	0.5	1	1.5		2	0	2	1	0.5	1.5	
6	—	—	—	—	—	—		—	—	—	—	—	—	
7	—	—	—	—	—	—		1	3.5	4.5	3	7.5	10.5	
8	1	0.5	1.5	0.5	1	1.5		1	1	2	1.5	0.5	2	
9	5	3	8	6	4	10		—	—	—	—	—	—	
10	2	4	6	1	1	2		3	3.5	6.5	—	3.5	3.5	
11	—	—	—	—	—	—		—	—	—	—	—	—	
12	—	—	—	—	—	—		—	—	—	—	—	—	
13	5.5	0	5.5	1.5	4	5.5		—	2.5	2.5	—	—	—	
14	—	—	—	—	—	—		—	—	—	—	—	—	
15	—	—	—	—	—	—		—	—	—	—	—	—	
			29.5			31	1.05			35.5			32.5	0.92

every tooth of each pair of twins, on each axis, and the derivation therefrom of the bilateral and unilateral differences. The measurements were made from the following loci:

Mesio-incisal angles of incisors
Apices of cusps of canines
Apices of buccal cusps of bicuspids
Apices of mesio-buccal cusps of molars

Table 31 shows the bilateral differences for the corresponding jaws of each pair of twins arrayed against the unilateral differences between the corresponding sides.

When the unilateral differences of either side are compared with the bilateral differences for each twin, there are four possible comparisons for the mandible, and four for the maxilla, on each co-ordinate, giving a total of 60 on each co-ordinate for each jaw for the series of fifteen pairs of twins.

In all these comparisons ULD should be less than BLD. The following summary represents the cases in which it is *not less*, and the frequency with which this occurs. Twins are quoted by number, and frequency is indicated by repetitions of the numerals.

Mandible								*Maxilla*							
X				*Y*				*X*		*Y*					
1	1			2				3		1	1				
2	2	3	3	3	3			4	4	2	2				
6	6	6	6	7				5	5	3	3				
7				8				6	6	4	4				
8	8			11				7		6	6				
9	9			13				8		7	7				
12								9		8	8				
12	13							10		9					
15	15							13		11	11	11	11		
								15		13	13	13	13		
										15					
20				7				13		24					

Hence out of a total of 240 comparisons (for X and for Y measurements on mandible and maxilla), there were 64 instances in which the expected relation was not found.

Physical conditions which might prove sources of error have been disregarded in this computation, on the ground that the errors involved would tend to vitiate rather than substantiate the hypothesis advanced. The effects of extracting teeth, restorations, and the total disregard of asymmetrical eruption and inverse symmetry, all tend to increase the unilateral differences. As many of these dentures are mutilated, there probably is a high degree of error to upset individual comparisons, and we will observe in succeeding and more general comparisons whether these individual exceptions, representing roughly 25 per cent of half-jaws, have offset the general result.

The embryological origin of identical twins, as previously portrayed, indicates that each pair is really an indiscerptible unity. Therefore, complete rather than unilateral comparisons should be made. As the lowest forms of life are unilateral organisms, every bilateral organism is in a sense a twin. A pair of identical twins may be conceived as a single quadrilateral organism which has fortuitously separated into two bilateral organisms, each part of a single system. Sometimes, indeed, they do not separate, and we have conjoined twins. For this reason, it is fairer to compare the sum of the bilateral differences with the sum of the left and right unilateral differences, as in *Table 32*, rather than to compare each of the two bilateral differences with each of the two unilateral differences.

Table 32 presents a comparison of total bilateral and unilateral differences for each jaw on each co-ordinate. Out of the total of 60 comparisons for the total of separate jaws, there were 10 instances in which ULD was larger than BLD, or 16.6 per cent.

Mandible	*Maxilla*
X	*Y*
6	4
8	6
15	7
3	8
	11
	13 13
	7

Table 33 shows the total BLD and ULD for each pair of twins on each co-ordinate. In five out of the 30 comparisons, ULD was larger than BLD.

X	*Y*
6	6
15	8
	11
2	3

In the totals of UL and BL differences, given in *Table 33*, it appears that the ULD on each co-ordinate is distinctly below the BLD. The actual ratios are:

X ULD : BLD = 0.749 : 1
Y ULD : BLD = 0.819 : 1

Table 34 represents the BLD and ULD grouped, in relation to the type of teeth for mandible and maxilla, separately on each co-ordinate for each pair of twins. The appended summary gives these data for mandible and maxilla together for the series of twins.

BLD and ULD

Tooth	*Mandible*		*Maxilla*	
	X	*Y*	*X*	*Y*
I	67.5	33.5	38.5	19
I^2	82.5	52	54	52
C	70	71	57	61.5
B	67.5	101	70.5	76
B^2	86	97.5	61.5	69
M	74.5	95.5	54.5	82.5
M^2	62.5	65	53	63.5

The number of the individual exceptions to the expected relation of BLD as greater than ULD may be grouped as follows (square-bracket enclosures represent the identificaton numbers of the twins):

Tooth	*Mandible*		*Maxilla*		*Total*	*Average per tooth*
	X	*Y*	*X*	*Y*		
I	0	0	1[9]	1[1]	2	0.016
I^2	1[15]	4[2,6 / 10,14]	5[1,3,8 / 10,13]	6[3,8,10 / 12,13,14]	16	0.133
C	0	4[8,10 / 11,15]	1[9]	3[1,9 / 10]	8	0.066
B	4[3,6,8 / 14]	0	6[3,9,11,12 / 13,14]	3[6,11 / 13]	13	0.108
B^2	5[2,3,8 / 11,14]	1[1]	1[6]	4[6,8 / 11,13]	11	0.091
M	1[6]	4[4,6 / 9,11]	2[11,13]	3[9,11 / 13]	10	0.083
M^2	2[3,13]	2[4,9]	3[4,7,8]	7[7]	8	0.066
					68	

The total is an index of the average frequency with which environmental conditions have upset developmental tendencies. The order of frequency is this: (1) I^2, (2) B, (3) B^2, (4) M, (5) C, (6) M^2, (7) I. However on account of the traumatized cases in this group, the results are unreliable. Indices of $\frac{\text{ULD}}{\text{BLD}}$, for maxilla and mandible separately, and also together, follow:

Tooth	*Mandible*		*Maxilla*		*Mandible and Maxilla*	
	X	*Y*	*X*	*Y*	*X*	*Y*
I	0.45	0.45	0.94	0.64	0.57	0.57
I^2	0.59	1.05	0.76	0.97	0.65	1.00
C	0.73	0.73	0.57	1.00	0.81	0.87
B	0.80	0.60	1.40	0.96	1.04	0.75
B^2	0.78	0.46	0.66	0.94	0.71	0.71
M	0.73	0.80	0.74	0.93	0.73	0.86
M^2	0.77	1.05	1.08	0.92	0.85	0.98

In only one instance does the index exceed 1 by an appreciable amount—that of the X co-ordinate of the maxillary first bicuspid.

It may be said that the lower the index, the less has been the environmental disturbance of developmental tendencies. Below is given the regrouping accordingly, for maxilla and mandible separately, and also together:

	Mandible		*Maxilla*		*Mandible and Maxilla*	
	X	*Y*	*X*	*Y*	*X*	*Y*
1	I	I	C	I	I	I
2	I^2	B^2	B^2	M^2	I^2	B^2
3	C	B	M	M	B^2	B
4	M	C	I^2	B^2	M	M
5	M^2	M	I	B	C	C
6	B^2	I^2	M^2	I^2	M^2	M^2
7	B	M^2	B	C	B	I^2

The *environmental effects* upon individual teeth in the fifteen pairs of twins may be summarized as follows:

(1) The central incisors, on the whole, suffer least from environmental conditioning, both in a lateral and antero-posterior direction, those of the maxilla being more variable in a lateral direction.

(2) The lateral incisor in general is more variable in the antero-posterior than the lateral direction.

(3) The canines in general are slightly more variable than molars in both antero-posterior and lateral directions, with a reverse relation for mandibular canines. Those of the maxilla are the least conditioned laterally and the most antero-posteriorly on the Y co-ordinate.

(4) The first bicuspid generally is the most variable tooth in a lateral direction, and less so than both molars and canines antero-posteriorly. However, it is less variable than the maxillary canine in an antero-posterior direction.

(5) The second bicuspids in general are less variable than both molars and canines, but more so than central incisors, and in a lateral direction. The mandibular second bicuspid is the most variable of these in a lateral direction, but less so than the first bicuspid. The maxillary second bicuspid is more variable than the canine in a lateral direction.

(6) The molars in general are less conditioned than canines, but more so than both central incisors and second bicuspids. The

mandibular molars are affected slightly more by environment than the mandibular canines; the maxillary molars, more in a lateral direction, but much less so in an antero-posterior direction than maxillary canines.

(7) The second molars in general show great variability in both lateral and antero-posterior directions, except the maxillary second molar, which is fairly stable in an antero-posterior direction.

In view of our total ignorance of the frequency and extent of incidence of inverse symmetry in the teeth of identical twins,[4] it was deemed inadvisable to make too close a scrutiny of pathological findings, as the results would have a doubtful significance. The following distinct evidences of inverse symmetry or "mirror-imaging" were found:

(1) In the x-ray shadowgraphs of the Behrens twins (*fig. 16*), it will be seen, from the prints of the maxillary bicuspid region, that there is a closer resemblance between the right of either and left of the brother, than between their corresponding sides, i.e., left like right and right like left, rather than left like left, and right like right. (These pictures were taken from the same angles, with the twins as nearly as possible in the same position.)

(2) In the McGraw twins, the last mandibular deciduous tooth retained in the mandible would seem homologous with the last one retained in the opposite side of the mandible of her sister (*fig. 14*). Both these teeth were retained distinctly longer than any of the other deciduous teeth.

(3) Although apparently symmetrical to visual examination, the Hewett twins will be seen, from their table of measurements, to offer a closer opposite-sided than a same-sided correlation (*fig. 15*).

(4) The Buchner twins present a similar type of malocclusion involving opposite sides of the jaws (*fig. 10*).

In conclusion, it may be said that the ratio of $\frac{\text{ULD}}{\text{BLD}} < 1$, established in the earliest stages of embryonic development for any character

[4] In order to reveal beyond question the workings of the inverse symmetry and asymmetry mechanisms in the fifty pairs of twins, and also to put on permanent record the pathological findings, x-ray shadowgraphs of the whole dentures of thirty pairs of identical twins were secured. The material is now under analysis.

in identical twins, is in the case of the dental arches maintained throughout the ages studied, i.e., 3.5 to 24 years.

IV. SUMMARY

In order to present, in generalized form, the relations of hereditary and environmental effects, as they appear concretely in the dentures of the fifteen pairs of twins examined, two sets of graphs were constructed, one representing composites of the X and Y measurements made for fifteen of the twins (*fig. 17*); another representing composites of the X and Y measurements for the fifteen co-twins (*fig. 18*). This undertaking involved the necessity of dividing the twins into two groups; one, designated A, containing one of each pair of twins; the other, designated B, containing the second of each pair of twins. It was aimed in this way to distribute differences between co-twins as evenly as possible in the two groups.

The assignment of the twins to these two groups was accomplished in the following manner (*fig. 17*): The total of the X measurements for each twin was found, and the pairs of twins were put in array. The first, fourth, fifth, eighth, ninth, twelfth, thirteenth, sixteenth, seventeenth, twentieth, twenty-first, twenty-fourth, twenty-fifth, twenty-eighth and twenty-ninth, were assigned to the A group. The second, third, sixth, seventh, tenth, eleventh, fourteenth, fifteenth, eighteenth, nineteenth, twenty-second, twenty-third, twenty-sixth, twenty-seventh, and thirtieth were assigned to the B group. The average X and average Y measurements for each tooth, for each group as a whole, were calculated (*Table 35*), and these measurements were plotted for the lower arch of the A group to form a graph in the shape of an arch (*fig. 18*), with a similarly constructed arch for the B group superimposed upon it. A similar procedure was carried out for the upper arch. The degree of correspondence in the superimposed arch forms, and the coincidence of cusps, are measures in a general way of the extent to which the thesis here maintained is supported, when the entire number of twins examined is considered.

Hence, the positions into which the teeth erupt are not determined by the allocation of a kind of "no-man's land" between opposing physical forces, where the inward pressure of the lips and cheeks neutralises the outward pressure of the tongue. Except in individual

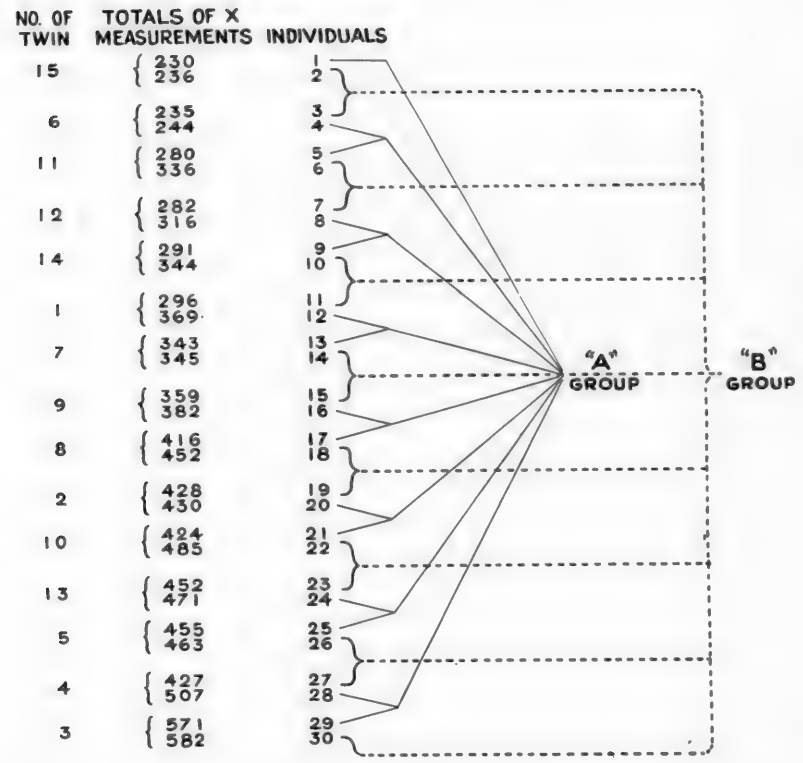

FIG. 17. GRAPH SHOWING COMPOSITES OF THE X AND Y MEASUREMENTS FOR FIFTEEN TWINS

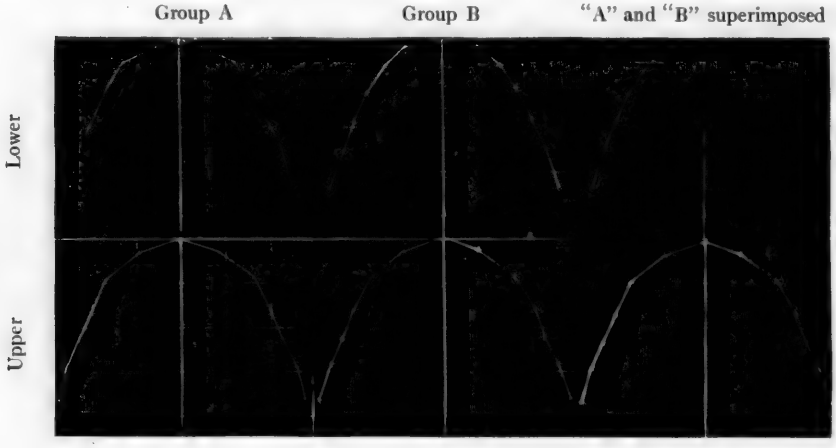

FIG. 18. GRAPHS SHOWING COMPOSITES OF THE X AND Y MEASUREMENTS FOR FIFTEEN CO-TWINS

cases where abnormal habits have interfered with intrinsic forces, the arch form, previsioned in the germ cells, is carried to fruition in the adult, and there is very little justification to assume that environment, either intra-oral or extra-oral, under average conditions in life, plays a very great rôle. Therefore, every interference with the general form of the dental arch may be violating and opposing inherent forces, and our degree of success may depend merely upon the elasticity of Wolff's Law, which says in effect that the growth of bone depends upon the need for it.

This may serve to explain some not infrequent and baffling failures, in the nature of reversions, which confront us subsequent to those

TABLE 35

Average X and average Y measurements for each tooth for each "A" group and "B" group as a whole

TOOTH	UPPER				LOWER			
	A		B		A		B	
	X	Y	X	Y	X	Y	X	Y
I	0.37	0.27	0.35	0.38	1.02	0.22	0.87	0.27
I^2	8.70	2.97	85.67	3.08	5.02	1.87	8.15	1.85
C	16.43	9.00	16.18	9.07	12.46	6.05	12.46	6.05
B	20.05	16.32	20.13	16.07	16.70	11.93	16.70	11.95
B^2	22.75	22.45	22.86	22.70	19.41	18.22	19.87	18.31
M	25.68	29.18	25.29	28.29	22.04	23.81	22.17	25.00
M^2	27.25	36.31	28.08	36.43	24.86	32.86	25.47	33.61

corrective endeavors that aim at an "ideal" or "harmonious" arch form. The stimulation towards development, artificially created by an appliance, may disappear with the removal of that appliance. It does disappear, at least in some cases; and, until the proportion of these to the *whole* number of successfully treated cases in that group is determined, it is incumbent on us to be more conservative in every way in treatment. For it can no longer be said that with mechanical appliances we are aiding nature to attain an ideal condition which, unaided, she could not produce on account of some extrinsic developmental handicap of epigenetic origin. In the mass of the population the arch form is inherited, and orthodontic interferences purporting to

change this developmental limitation set by heredity fall into the category of surgical intervention, and find their justification or refutation on the same grounds that such procedures do, namely:

(1) The possible value of the intervention, and its possible dangers.

(2) The chances of success, computed on the basis of the *whole* number of treated cases.

(3) The demonstration of a distinct extrinsic or epigenetic cause, which can be counteracted.

The elucidation of these questions devolves upon every individual who practises orthodontia, and it is his bounden duty to justify his procedures. It is not an obligation (as stressed in a recent editorial challenge entitled "We approach an era of extraction") of the geneticist to show which cases are inherited or what the heredity limits for a given case are: the onus is rather on those who practise orthodontia, to justify their procedures in a manner acceptable to statisticians, or to differentiate genetic and epigenetic factors in individual cases.

This brings up anew the old requisite of scientific orthodontic procedure; namely, a classification based upon etiology of types of cases and criteria for distinguishing individual cases. The possibility and method of achieving such a classification has been revealed in this essay. If this modest contribution serves to stimulate increasing contributions to our literature along those lines the costly and time-consuming efforts of the author will have been amply requited. It is manifestly a greater achievement to practise orthodontia in a manner calculated to eliminate reversions, with the goal of an optimum state for the *individual* in mind, than to strive after a preconceived ideal in every case. The condition of individual optimum does not coincide necessarily with a collective ideal.

The accumulation of the necessary data upon which to found a classification based on etiology, differentiating primarily between extrinsic and intrinsic forces, is one in which the whole profession could participate. If every practitioner merely reported carefully to the literature the identical twins in his practice or acquaintance, sufficient data would be available to found this classification, not only for types of cases but also for individual teeth in three dimensions of space, and to secure criteria for distinguishing in individual cases extrinsic from intrinsic causes of mal-occlusion. In this direction

lies our greatest hope of divorcing empiricism from the practice of orthodontia—through a method which constitutes, so to speak, a flank attack upon problems that have defied solution by frontal attack through the orthodox channels of dental research.

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HOW DENTAL STATISTICS ARE SECURED IN THE METROPOLITAN LIFE INSURANCE COMPANY[1]

THADDEUS P. HYATT, D.D.S., F.A.C.D.

WITH AN ADDENDUM BY ALFRED J. LOTKA, M.A., Sc.D.

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We have come to recognize as an established line of research the development of operative procedure for the prevention of decay, with special attention to the study of specific locations of vulnerable or diseased surfaces. For the prosecution of this line of research we should be able to derive valuable information from a careful study of recorded findings of the frequency of caries in the several surfaces of the different teeth. This means that we need a systematic statistical service to gather data, to organize them, tabulate them systematically, and, by the proper statistical technique, to draw correct conclusions from them.

In view of the importance of dental statistics for the etiological study of dental caries, it is highly desirable that careful consideration be given to the procedure which is followed in securing these figures. The first condition for effective investigation is, of course, to see that the examinations are made thoroughly and recorded carefully and correctly. When the dental condition of a sufficiently large group of persons has been thus recorded, and correct procedure in the statistical treatment of the findings has been followed, then the information obtained and properly presented (for example in the form of graphs) will aid us in determining to what extent dental caries can be controlled or prevented. It is desirable that all who are interested in this subject be informed regarding the various

[1] Read at a meeting of the New York Section of the International Association for Dental Research, Columbia University Club, 4 West 43rd St., New York City, February 29, 1928; also at a session of the Sixth Annual Meeting of the International Association for Dental Research, Hotel Mayflower, Washington, D. C., March 25–28, 1928. See abstract: *Journal of Dental Research*, 1928, viii, p. 413.

steps by which the statistical figures are secured, recorded, transferred to cards for mechanical tabulation, then tabulated and finally presented to the eye in the form of graphs or charts. The first and most important step is to evolve a correct and simple procedure for examining and recording the actual and complete dental condition present, at the time the examination is made. I shall

FIG. 1. View of the Clinic, with twelve chairs. In the first booth a dental hygienist is making an examination, and a clerk is recording findings.

now describe, step by step, the process followed in collecting and working up the statistical data that become available in the course of the work of the Dental Division at the Home Office of the Metropolitan Life Insurance Company.

Fig. 1 gives a view of our Clinic with twelve chairs. There are ten dental hygienists, and one full-time and two part-time dentists.

The first booth shows a dental hygienist making an examination and one of the clerks recording the findings.

Fig. 2 shows a mouth mirror and an explorer used in making examinations. We have found an explorer of this shape the most practical, because most universal in its application. It is needless to add that these explorers must be constantly sharpened.

Fig. 3 shows the outside covers of our primary record chart, which folds at the top. The front and the back covers are the same.

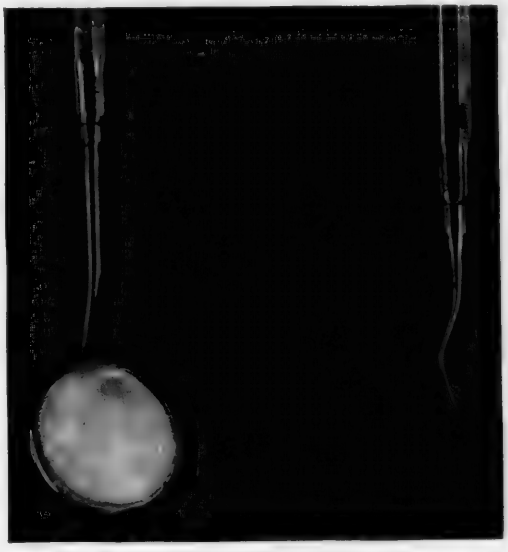

FIG. 2. Mouth mirror, and an explorer, used in making examinations. Actual size, with most of each handle cut from the illustration.

As examinations are made twice each year, each chart is good for eight years. One such chart is kept for each person examined. In our Clinic the teeth are numbered from the patient's right to his left, starting with the upper right third molar and ending with the lower left third molar. The teeth are numbered this way so that the recording and reading of the records may follow the usual manner of reading and writing: from left to right, the reverse, of course, of their order in the patient's mouth.

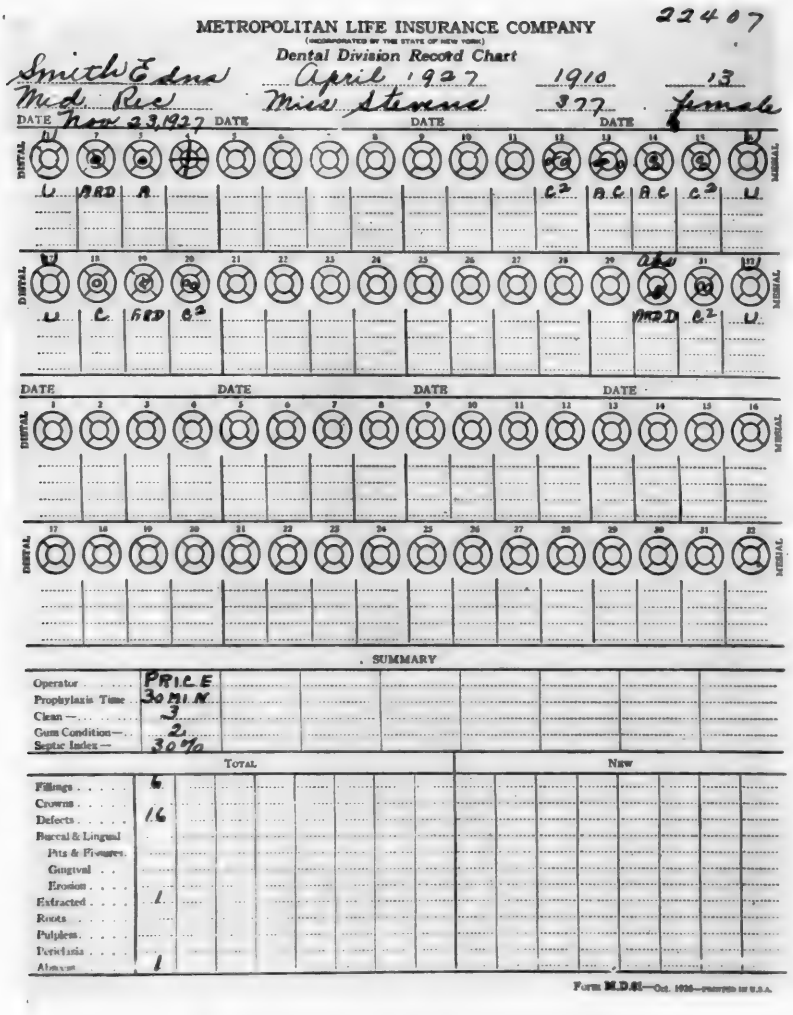

FIG. 3. Outside cover of the primary record chart

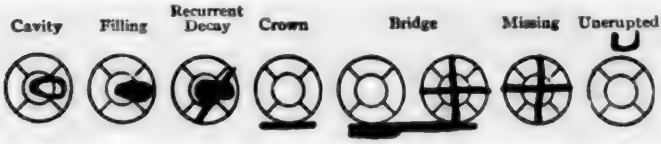

FIG. 4. Symbols used in recording data

X-RAY RECORD

Form M.D.61 A

DATE	No Films	Missing	Unfilled	Incompletely Filled	Completely Filled	Infection	Abscess	Root	Unerupted	Impacted	Pericasia	REMARKS

FIG. 5. Inside of back cover of the "primary record chart" shown in *fig. 3.* Used to record x-ray findings. The illustration shows only the heading and two of the 50 blank lines for entries.

EMERGENCY

Form M.D.

DATE

FIG. 6. Inside of front cover of the "primary record chart" shown in *fig. 3.* Used to record emergencies, consultations, etc. The illustration shows only the heading and two of the 36 blank lines for entries.

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32
	2nd P 17	2		10	4	14	4	4	1	5	1	1	5	1	6 4	4	5	10	10	2	2	4	3	4		10	1	1	1	1	1	1	1	3	4	2
1 - P	F 23	5	0	5 7	10	12	10	4 7	7	7	7	6	6		11	6 7	4		12	4	3		14	3	5 4	4	5	1	1	1	1	1	5		12	9	5	4
2 - U	M 19	2		19	4	4	4	1	5	1	5	1	1	5	4	4 5	4 5	2	2	4	4	1	1	1	1	1	1	1	1	1	1	1	3	3	4
3 X	F 24	4	5	10	8	10	9	3	6	7	5 7	6 7	5 7	6	6 7	12	3	5	10	5	10	2	4	5 4	3	7	10	4	1	1	1	1	1	1	4	3	4	4
4 - Occ	F 25	4	5		10	1	1	1	1	1	1	1	1	6	3	5 7	10	4	2	2	4	4	6	1	1	1	1	1	1	1	1	6	5	4	3

FIG. 7. Sheet used for the transfer of records from the "primary record charts" of the individual persons to summarize the findings for all the different persons examined. Shows only the first four lines of a specimen.

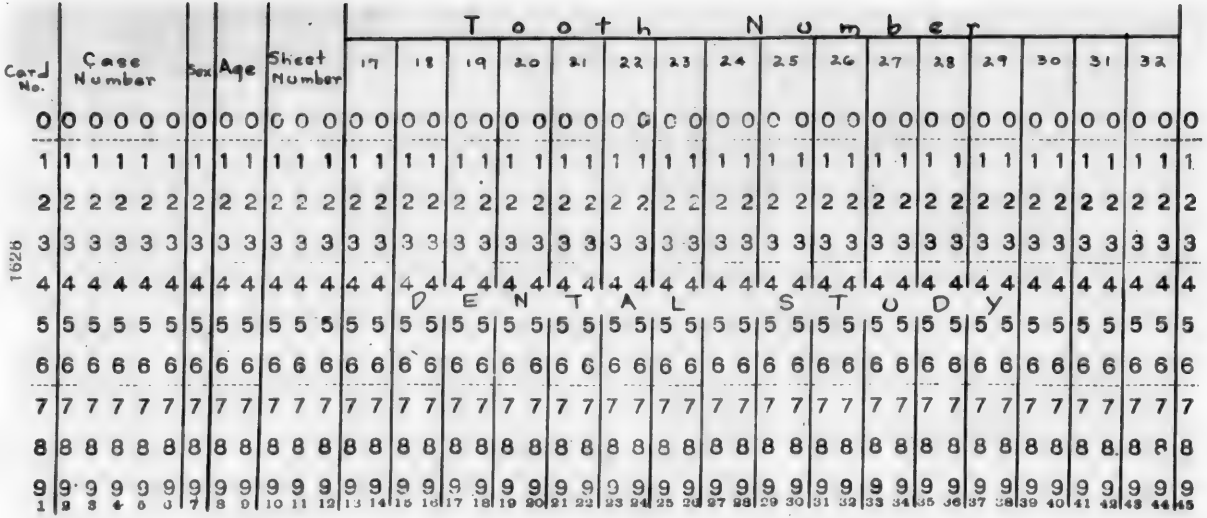

FIG. 8. Mechanical tabulator card ("punch card"). Form used for lower teeth (17–32)—duplicate of form used for upper teeth (1–16). Use shown in *fig. 9*.

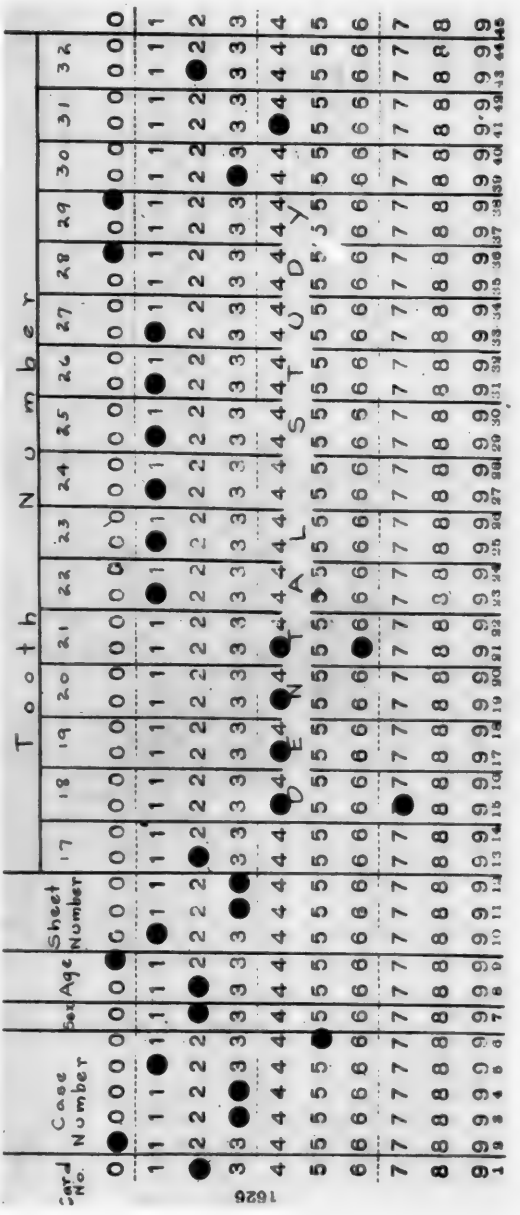

FIG. 9. Record on a mechanical tabulator card ("punch card"). Compare with *fig. 8*

Fig. 4 shows the symbols used in making records on the chart. As will be seen, circular symbols divided into five segments or fields are used, in preference to actual illustrations, to represent the thirty-two teeth in the human dentition, and the five surfaces on each. No median line has been drawn on the chart to separate

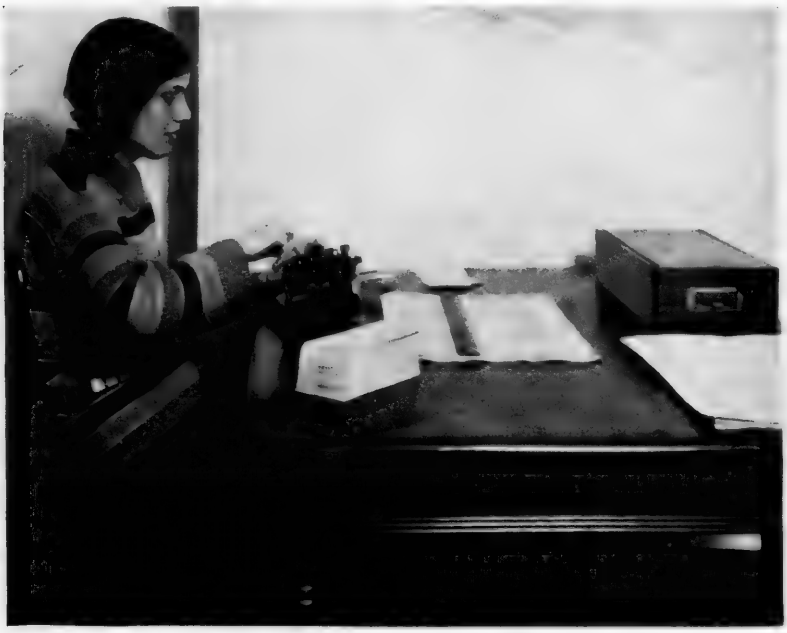

FIG. 10. One of the assistants punching a card (*fig. 9*). A ruler is used as a visual safeguard against mistake. See also *fig. 11*.

right from left. The particular chart here reproduced (*fig. 3*) gives the following information, among other things:

1—Upper right third molar; unerupted
2—Upper right second molar; occlusal amalgam filling with recurrent decay
3—Upper right first molar; occlusal amalgam filling
4—Upper right second bicuspid; extracted
12—Upper left first bicuspid; a distal and an occlusal carious cavity

13—Upper left second bicuspid; a distal-occlusal amalgam filling and a mesial carious cavity
30—Lower left first molar; lingual-occlusal amalgam with recurrent decay, devital and abscessed
31—Two occlusal cavities
32—Unerupted

FIG. 11. An assistant testing whether "punch cards" have been punched correctly. Duplicates, made by different assistants, are held together in the same position over a boxed light with an opal-glass front. If the holes do not exactly correspond, error is indicated.

The left hand compartment of each symbol represents the distal surface of the tooth; the right compartment always represents the mesial surface; the upper is buccal or labial, the lower lingual; while the central compartments represent the occlusal surface. It is therefore quite simple for a layman to read these charts with

very little instruction. The omission of the median line prevents the confusion that would otherwise be apt to arise through the necessity of reversing the names of the two compartments on the right and on the left, respectively, of the median line. This simple precaution thus practically eliminates the possibility of thirty-two different errors of recording.

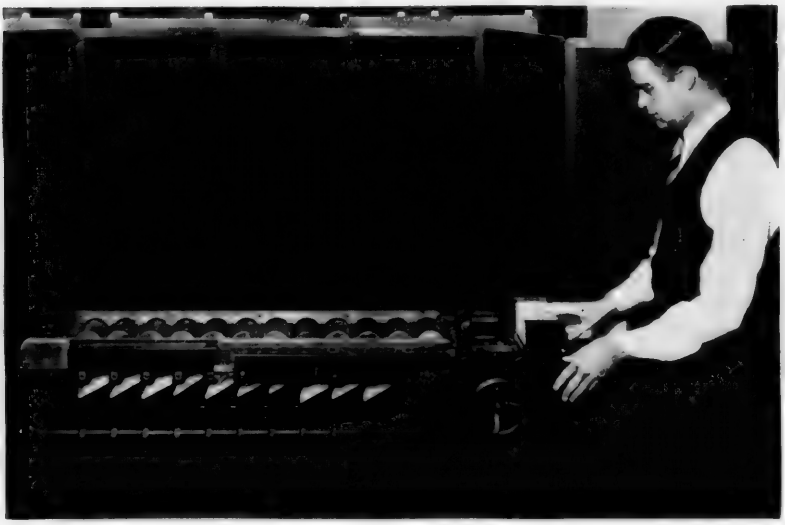

FIG. 12. An assistant using a counting and assorting machine set to pick out and count certain data on "punch cards."

Fig. 5 shows the inside of the back cover of the primary record chart used to record x-ray findings. *Fig. 6* shows the inside of the front cover, which is used to record emergencies, consultation, etc.

Fig. 7 shows the sheet used for the transfer of records from the primary record charts of the individual person to summarize the findings for all the different persons examined. The numbers in the heading correspond to the tooth numerals on the primary chart. Down the left side are shown the code numbers used to indicate tooth conditions. Inasmuch as it is important to avoid errors, definite information must be given for each tooth with no excep-

tions; so 1-P stands for *perfect*, i.e., present in the mouth, tooth showing no cavities, no fillings, no devital pulp. Similarly the following code numbers designate the conditions stated:

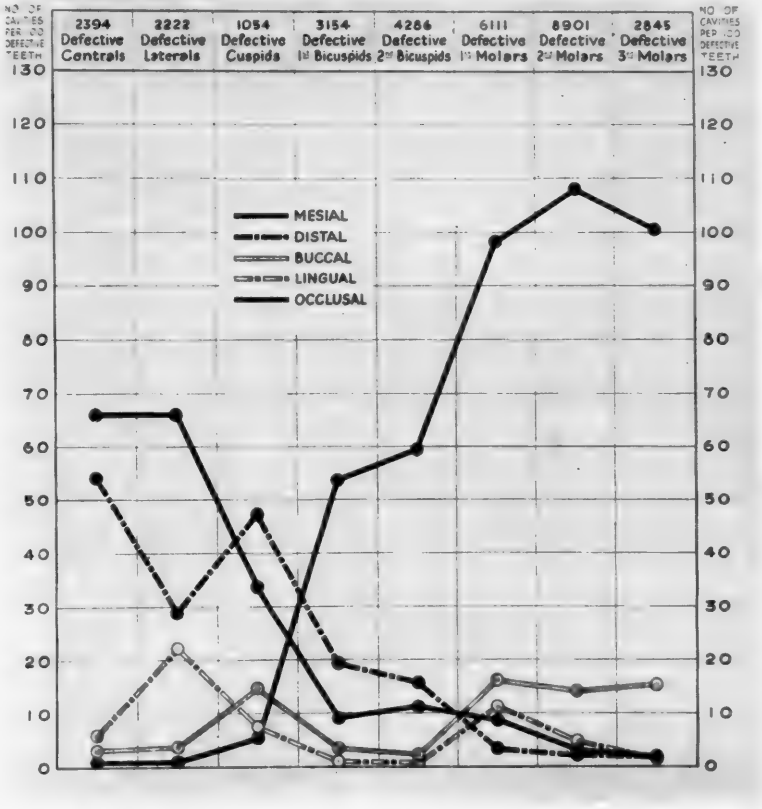

FIG. 13. Graph showing defective surfaces found in dental examinations of 2943 men and women between 16 and 25 years of age. There were 30,967 defective teeth, exclusive of missing and badly decayed teeth, roots, and crowns.

2—U; unerupted
3—X; extracted
4—Occ.; occlusal cavity or filling

Reading the first line we find the record has been taken from sheet

number 276, the patient a female, age 17. The upper right third molar is unerupted, the second molar has two occlusal defects (fillings being considered the same as unfilled carious cavities). The

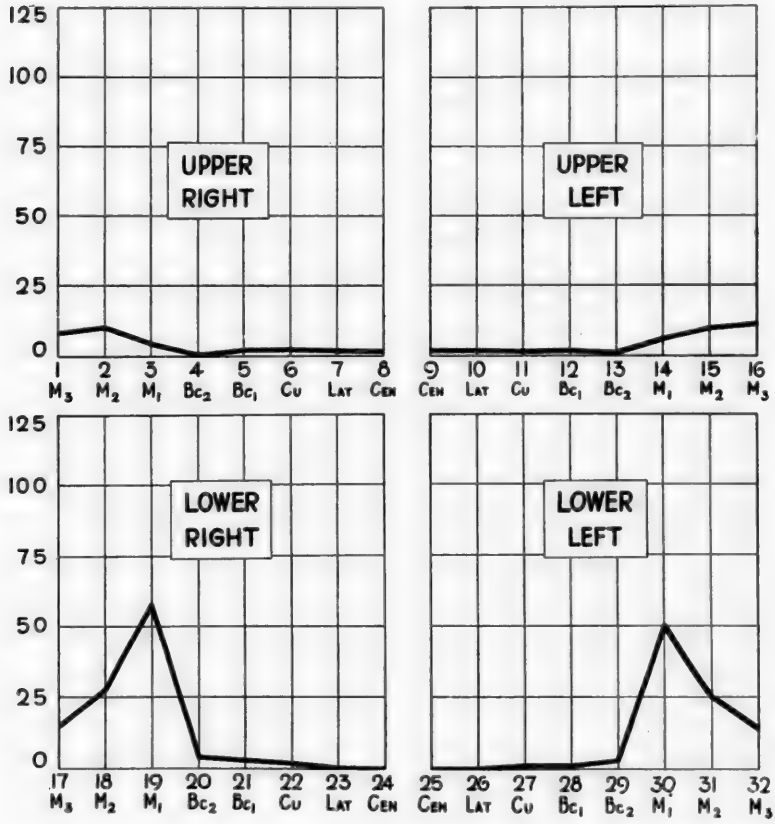

FIG. 14. Graph showing susceptibility of teeth to decay. Cavities in *buccal surfaces*, per 100 erupted teeth of specified kinds. Proportionate allowance has been made for missing or badly decayed teeth. See also *figs. 15–18.*

first molar has an occlusal defect and a compound lingual-occlusal defect. The rest of the line can be easily read and understood.

So far in this series the clerical work is carried out by the Dental Division. These sheets, when filled, are turned over to the

Statistical Department, where the information is then punched on mechanical tabulator cards, one of which is shown in *fig. 8*. Two are used for each patient, one for the upper teeth and one for the

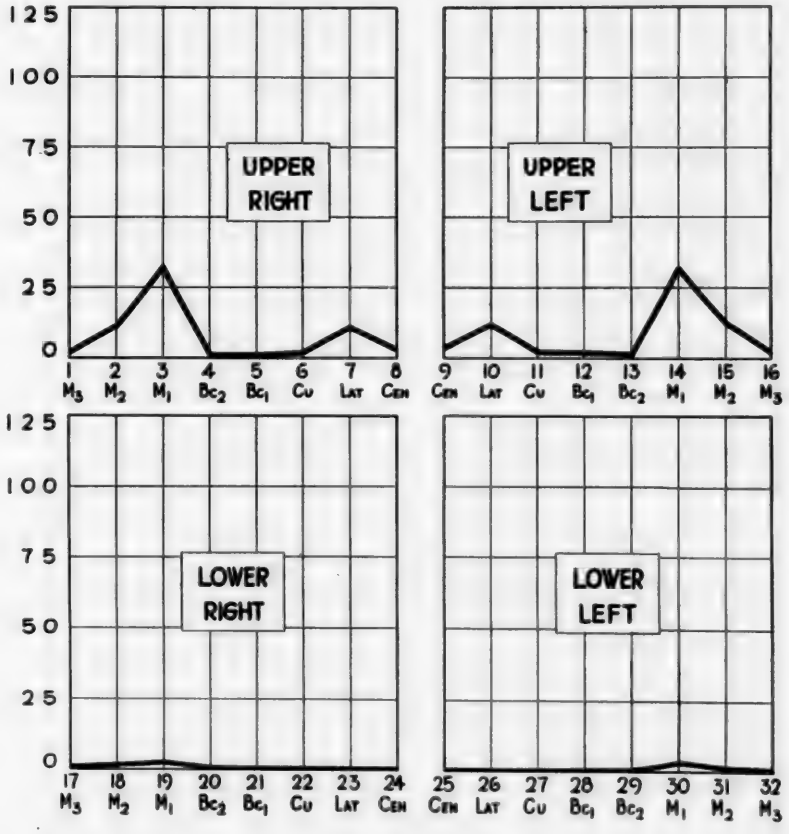

FIG. 15. Graph showing susceptibility of teeth to decay. Cavities in *lingual surfaces*, per 100 erupted teeth of specified kinds. Proportionate allowance has been made for missing or badly decayed teeth. See also *figs. 14* and *16–18*.

lower. Each card is made out in duplicate, so that there are in all four for each patient; see the description of *fig. 11* (p. 424). *Fig. 9* shows one of the cards punched. The first hole, in 1 under "card number," shows that this card gives the record for the upper teeth.

Other punches indicate case no. 3315; sex, female; age, 20; record taken from sheet no. 133. *Fig. 10* shows one of the girls punching the cards from the sheet. A ruler is laid along the line as a safety

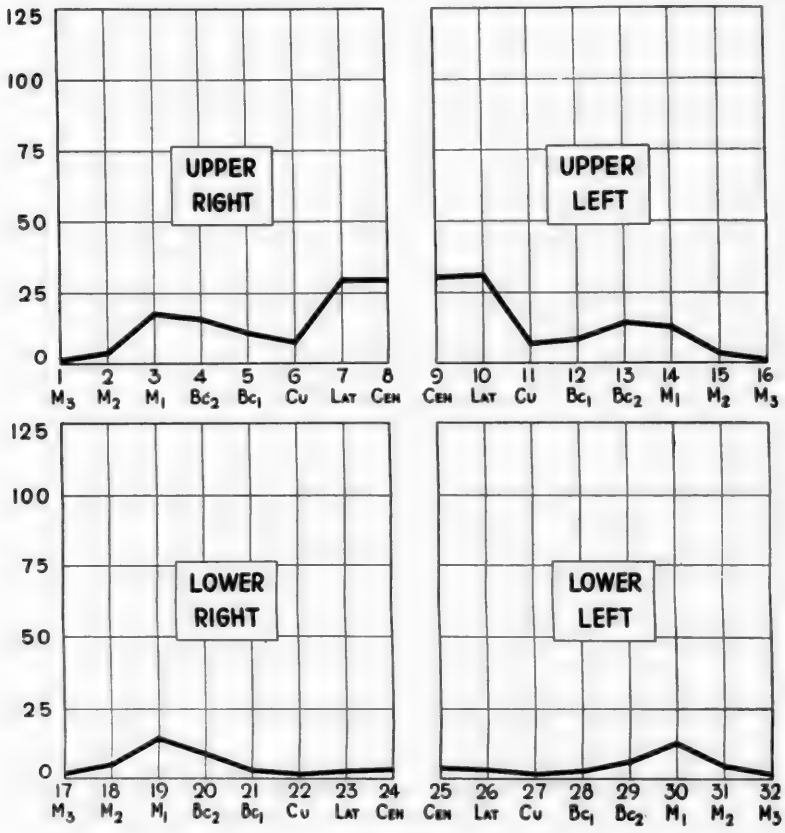

FIG. 16. Graph showing susceptibility of teeth to decay. Cavities in *mesial surfaces*, per 100 erupted teeth of specified kinds. Proportionate allowance has been made for missing or badly decayed teeth. See also *figs. 14–15* and *17–18*.

measure against copying a figure from a wrong line and thus making a mistake.

Fig. 11 shows the method of testing whether cards are punched correctly. Two are held together over a boxed light with an opal

glass front. As mentioned above, duplicate sets of cards, one white and one red, are punched by different operators for each patient. When duplicate cards are held before the light together, in the same position, all holes must correspond.

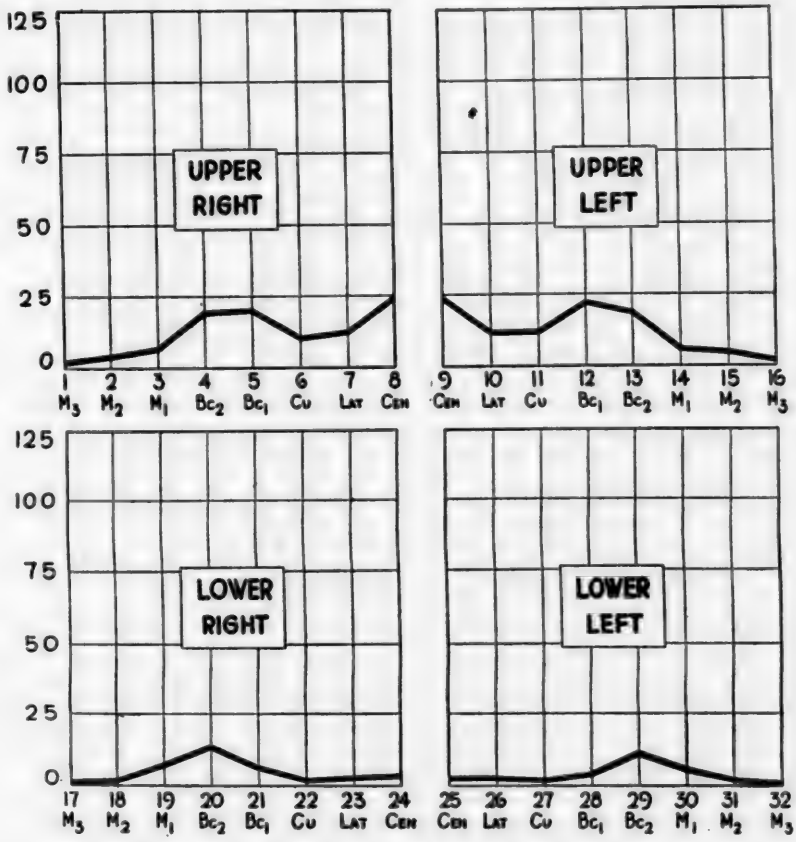

FIG. 17. Graph showing susceptibility of teeth to decay. Cavities in *distal surfaces*, per 100 erupted teeth of specified kinds. Proportionate allowance has been made for missing or badly decayed teeth. See also *figs. 14–16* and *18*.

Fig. 12. A stack of these punched cards are now placed in a counting and sorting machine, which has been set to pick out and count certain data. As the cards pass toward the end of the

machine, they are sorted and counted mechanically, each card falling into its proper compartment according to the holes punched in it. To find the number of persons having a mesial cavity in

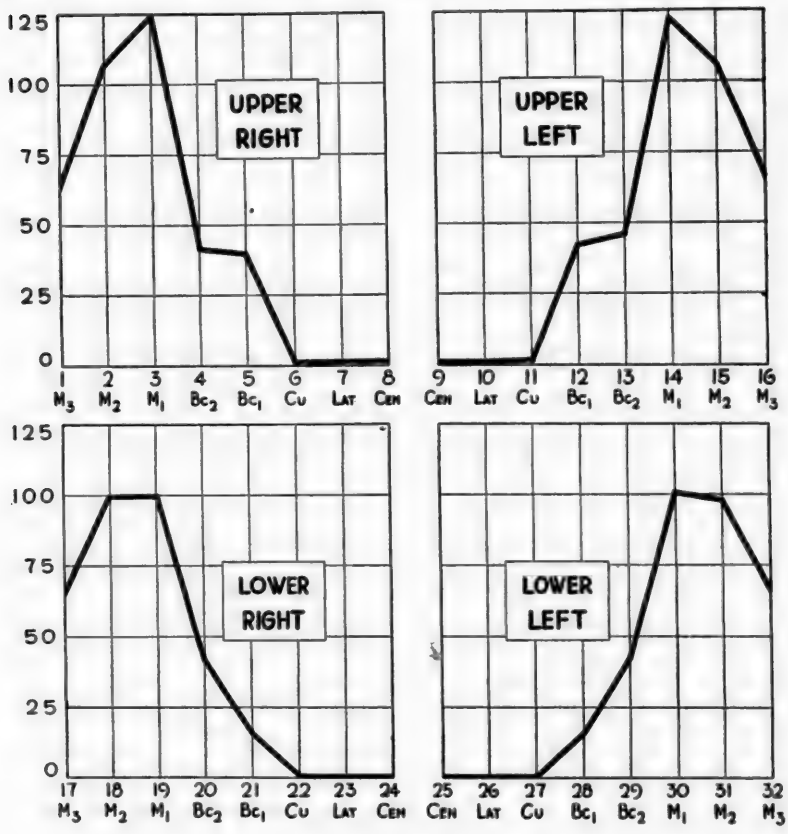

FIG. 18. Graph showing susceptibility of teeth to decay. Cavities in *occlusal surfaces*, per 100 erupted teeth of specified kinds. Proportionate allowance has been made for missing or badly decayed teeth. See also *figs. 14–17*.

tooth no. 4, for example, the machine is set for column 19 (see numbering of columns at bottom of *fig. 8*), and in the seventh compartment or box are received all those cards that have been punched 7 in the column selected, that is to say 19. A dial on this

box automatically registers the number of cards that have been dropped into it. The results of this machine-sorting are next recorded on a sheet as shown in *Table 1*. This completes the mechanical work upon the observed data.

The results are then sent to the mathematical section, where they are examined, construed, and elaborated into tables or graphs, which enable one to visualize and understand what significance may be attached to the numerical data obtained. *Fig. 13* shows one of the

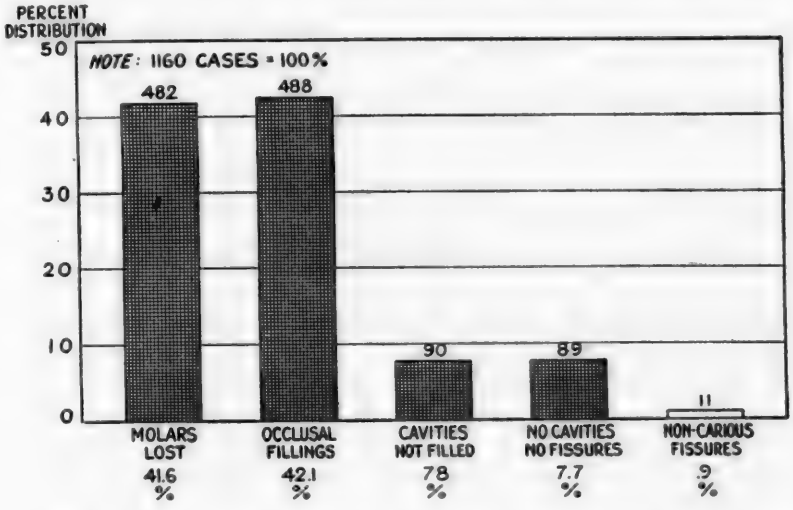

FIG. 19. Graph showing distribution of non-carious fissures in 1160 first and second molars, in adults between 30 and 40 years of age.

graphs made from a study of the dental condition of 2,943 young men and women between the ages of 16 and 25. It exhibits the number of cavities *per 100 defective teeth* found in the several surfaces of the teeth characterized in the boxes at the top of the diagram. So, for example, on an average, every 100 central teeth that were defective in any way, had 68 mesial cavities. This statement relates to all four centrals combined, and it should be understood in this statement that *perfect* teeth and *missing* teeth are excluded from the reckoning.

It is interesting to note the low average of mesial and distal defects in the molars as compared with the buccal and lingual defects. When these results were first presented to me, I questioned their accuracy regarding mesial and distal susceptibility of the molars, and requested a review of the work to ascertain whether mistakes had been made. It required about six weeks to go back and check up every step of the work. When I was informed that these figures were correct, I showed the graph to Professor Bödecker

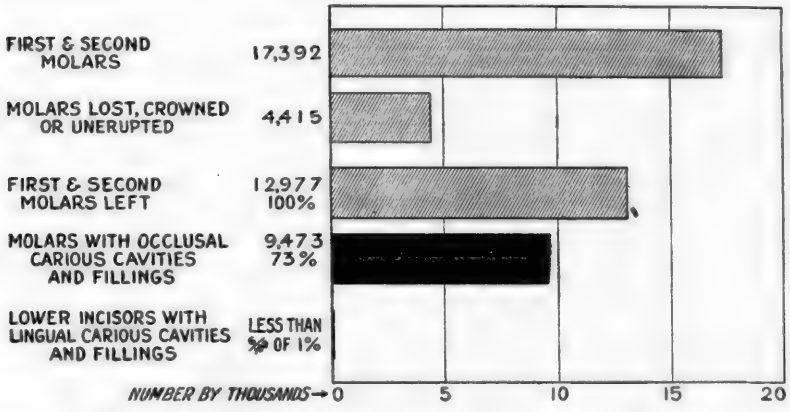

FIG. 20. Graph showing number of carious cavities in the lingual side of the lower incisors, compared with those in the occlusal side of the molars. Examination of 2174 women between the ages of 17 and 25 years. There are 1216¾ times more occlusal carious cavities in the molars than lingual carious cavities in the lower incisors.

of Columbia University without making any comment. After a few moment's study, Professor Bödecker looked up and exclaimed: "Have you noticed this?", and called my attention to the low average of the mesial and distal defects in the molars. On my assuring him of their correctness, Professor Bödecker visited our Clinic and made several examinations. Finding them remarkably correct, he urged on me the importance of inviting dentists to visit the clinic and make similar examinations, and to permit their names to be published so as to assure the profession at large of the accuracy of these statistics. This advice has been followed, and the members

of our profession named below have permitted the publication of their names in connection with this work:

Dr. W. A. Beatty	Dr. A. S. Litten
Dr. Charles F. Bödecker	Dr. B. A. Seam
Dr. E. H. Brophy	Dr. J. Shaffer
Dr. Connelly	Dr. O. J. Sorenson
Dr. Martin Dewey	Dr. F. L. Stanton
Dr. C. R. Ellis	Dr. A. B. Stark
Dr. J. Hanks	Dr. Alfred Walker
Dr. J. Kauffman	Dr. Maurice William

To make certain that proximal caries had not been overlooked, upward of 688 teeth or 1,376 proximal surfaces, which had not been recorded as having distal or mesial caries, have been very carefully x-rayed by the Rapier procedure. The result of this supplementary examination showed that only 1.25 per cent of caries had been missed. This small number cannot in any way change the ratio of caries susceptibility of these surfaces in comparison with other surfaces.

Figs. 14, 15, 16, 17 and *18* show, in a different way, some of the outstanding facts revealed by our investigation. They relate, respectively, to the buccal, lingual, mesial, distal, and occlusal surfaces. In each *fig.* (*14–18*) data for the thirty-two teeth of the dentition are shown seriatim from left to right, in two tiers, one for the upper jaw and one for the lower. A vertical line is drawn for each tooth on such a scale as to represent, by its vertical height, the susceptibility of each tooth and tooth surface to decay. For example, in the figure relating to buccal surfaces (*14*), tooth number 19 (lower right first molar) is represented by a line fifty-two units high. This means that, in every 100 lower right first molars, there is an average of 52 cavities; and so on for the other teeth. The graphs otherwise are self-explanatory.

The data represented in *figs. 14–18* are given numerically in *Table 2*. In the preparation of this table the following special allowances were made:

(1) Correction for missing teeth.

(2) Cavities classed as "compound with occlusal" were distributed pro rata among lingual, buccal, mesial, and distal surfaces.

(3) The figures for "all surfaces" (decayed) were added to each of the figures found for the separate surfaces.

TABLE 1 (in two parts)

Conditions of teeth. Based upon examination of 802 white females. Ages 25 and less

First part: upper teeth

CODE	CONDITIONS	TOOTH															
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
	Total conditions	839	954	1063	916	889	831	937	958	966	935	836	895	909	1048	991	853
P	Perfect	115	104	63	362	384	638	466	437	434	444	605	363	350	80	108	130
U	Unerupted	437	—	—	3	1	3	6	—	1	7	7	1	3	—	—	429
X	Missing	41	36	189	58	73	20	33	20	23	35	22	65	76	176	42	36
O	Occlusal	166	365	239	144	108	4	5	—	1	2	4	138	161	233	370	166
B	Buccal	25	41	15	3	6	9	11	8	8	11	14	8	9	16	52	35
D	Distal	7	25	20	77	82	56	79	196	183	103	77	94	79	25	19	6
M	Mesial	6	11	55	62	37	41	205	213	227	195	53	42	69	54	23	2
L	Lingual	8	56	104	6	1	9	60	16	14	66	14	2	3	102	63	6
C	Crown	1	17	17	32	25	15	16	21	12	16	10	23	29	20	7	2
R	Root	1	—	15	9	3	—	—	1	—	1	—	2	4	9	2	—
S	Devital	—	5	18	18	15	1	22	22	26	19	2	10	11	21	6	—
	Badly decayed	1	8	10	3	4	—	—	—	—	—	—	7	3	7	4	3
W	All surfaces	—	4	4	1	1	—	3	1	1	1	1	—	1	8	8	1
T	Mesial-occlusal-distal	—	1	3	11	8	—	—	—	—	—	—	9	9	6	2	—
O*	Two occlusal or more	19	179	156	4	9	—	—	—	—	—	—	9	4	164	186	25
D-O	Distal-occlusal	1	30	10	46	89	3	1	—	5	—	2	86	46	18	28	1
M-O	Mesial-occlusal	2	11	58	65	36	1	2	2	3	—	1	27	48	48	17	3
B-O	Buccal-occlusal	—	12	7	2	1	—	—	—	—	—	—	1	4	8	3	
L-O	Lingual-occlusal	5	30	61	3	1	1	2	—	—	—	1	—	1	36	31	3
B-D	Buccal-distal	4	7	2	2	x 1	1	—	2	2	4	—	3	—	1	5	—
B-M	Buccal-mesial	—	—	1	2	—	2	5	1	—	5	2	—	—	2	1	—
L-D	Lingual-distal	—	3	3	1	1	23	5	7	12	5	16	1	—	1	2	—
L-M	Lingual-mesial	—	1	5	—	—	2	15	8	8	18	3	—	—	6	—	—
D-B-O	Distal-buccal-occlusal	—	4	—	1	1	—	—	—	—	—	—	2	—	3	4	2
D-B-L	Distal-buccal-lingual	—	1	—	—	—	1	—	2	2	1	—	—	—	—	1	—
M-B-L	Mesial-buccal-lingual	—	—	—	—	1	1	1	1	3	—	—	—	—	—	—	—
D-L-O	Distal-lingual-occlusal	—	3	5	—	—	—	—	—	—	—	2	1	—	5	1	—
M-B-O	Mesial-buccal-occlusal	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—
B-M-O-L	Buccal-mesial-occlusal-lingual	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—
M-O-L	Mesial-occlusal-lingual	—	—	1	1	1	—	—	—	1	1	—	1	—	2	—	—
B-D-O-L	Buccal-distal-occlusal-lingual	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
M-L-D	Mesial-lingual-distal	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
M-B-D	Mesial-buccal-distal	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
D-M-O-L	Distal-mesial-occlusal-lingual	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
B-O-L	Buccal-occlusal-lingual	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—
M-O-D-B	Mesial-occlusal-distal-buccal	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	

TABLE 1 (in two parts)

Conditions of teeth. Based upon examination of 802 white females. Ages 25 and less

Second part: lower teeth

CODE	CONDITIONS	TOOTH															
		17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32
	Total conditions	859	1003	1007	867	837	816	813	819	817	814	804	821	817	985	976	844
P	Perfect	115	31	28	393	628	752	750	750	753	755	754	656	463	33	37	128
U	Unerupted	379	2	—	2	1	—	—	2	—	1	—	1	1	—	—	386
X	Missing	38	86	368	71	12	2	4	—	2	2	4	14	47	390	98	32
O	Occlusal	200	477	213	182	79	2	—	—	—	—	1	60	179	203	483	210
B	Buccal	48	130	111	14	13	11	4	2	2	1	5	12	8	104	112	36
D	Distal	—	11	18	53	34	24	25	31	25	21	11	17	44	18	10	1
M	Mesial	5	28	31	24	18	17	24	24	30	28	16	10	30	29	26	2
L	Lingual	1	10	3	—	—	—	2	—	—	1	2	2	1	10	11	3
C	Crown	3	26	25	19	4	—	—	2	—	—	1	4	8	28	21	5
R	Root	—	2	17	—	1	—	—	—	—	—	—	1	2	11	2	—
S	Devital	1	17	34	7	2	2	1	3	—	1	1	2	4	21	11	—
	Badly decayed	2	4	11	4	1	—	—	—	—	—	—	—	3	12	5	1
W	All surfaces	1	5	9	—	—	1	1	—	—	—	—	—	1	5	2	—
T	Mesial-occlusal-distal	—	6	13	2	—	—	—	—	—	—	—	1	2	8	3	—
O[3]	Two occlusal or more	47	106	42	21	5	—	—	—	—	—	—	5	14	39	90	27
D-O	Distal-occlusal	—	17	17	53	24	1	—	1	2	1	—	24	41	16	10	2
M-O	Mesial-occlusal	5	14	24	13	8	1	—	—	—	—	—	6	11	22	20	1
B-O	Buccal-occlusal	5	10	11	1	1	—	—	—	—	—	1	—	2	11	13	6
L-O	Lingual-occlusal	1	9	10	3	1	—	—	—	—	1	—	—	2	9	12	1
B-D	Buccal-distal	2	—	—	2	1	—	1	2	—	1	1	1	6	6	—	1
B-M	Buccal-mesial	2	1	1	—	2	1	—	—	1	—	—	2	3	1	—	—
L-D	Lingual-distal	—	2	3	—	—	—	1	—	2	1	3	1	1	1	2	—
L-M	Lingual-mesial	1	1	1	1	1	1	—	2	—	—	4	1	2	—	1	—
D-B-O	Distal-buccal-occlusal	1	2	6	1	—	—	—	—	—	—	—	—	—	5	1	2
D-B-L	Distal-buccal-lingual	—	2	1	—	—	—	—	—	—	—	—	—	1	—	—	—
M-B-L	Mesial-buccal-lingual	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
D-L-O	Distal-lingual-occlusal	1	2	6	—	1	—	—	—	—	—	—	1	1	2	2	—
M-B-O	Mesial-buccal-occlusal	—	1	1	—	—	1	—	—	—	—	—	—	—	1	1	—
B-M-O-L	Buccal-mesial-occlusal-lingual	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
M-O-L	Mesial-occlusal-lingual	—	—	—	1	—	—	—	—	—	—	—	—	—	—	1	—
B-D-O-L	Buccal-distal-occlusal-lingual	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
M-L-D	Mesial-lingual-distal	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
M-B-D	Mesial-buccal-distal	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
D-M-O-L	Distal-mesial-occlusal-lingual	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
B-O-L	Buccal-occlusal-lingual	1	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—
M-O-D-B	Mesial-occlusal-distal-buccal	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

TABLE 2

Susceptibility of teeth to decay. Examination of 2,943 males and females under 25 years of age. Cavities per 100 erupted teeth (corrected for "missing" teeth)

TEETH	LINGUAL	BUCCAL	MESIAL	DISTAL	OCCLUSAL
Centrals					
Teeth 8, 9, 24, 25	1.52	0.80	16.58	13.55	0.81
Teeth 8, 9	3.03	1.49	29.72	24.09	1.31
Teeth 24, 25	0.02	0.11	3.46	3.02	0.30
Tooth 8	2.67	1.35	29.16	24.08	1.54
Tooth 9	3.40	1.65	30.27	24.08	1.09
Tooth 24	—	0.04	3.23	3.42	0.33
Tooth 25	0.04	0.19	3.68	2.62	0.26
Laterals					
Teeth 7, 10, 23, 26	5.50	0.97	16.33	7.14	0.72
Teeth 7, 10	11.03	1.61	30.02	11.66	1.27
Teeth 23, 26	0.11	0.31	2.82	2.56	0.17
Tooth 7	10.42	1.69	29.13	11.90	1.34
Tooth 10	11.65	1.53	30.93	11.42	1.19
Tooth 23	0.13	0.34	2.62	2.87	0.16
Tooth 26	0.08	0.29	3.00	2.26	0.19
Cuspids					
Teeth 6, 11, 22, 27	0.97	1.93	4.27	6.19	0.94
Teeth 6, 11	1.83	1.97	7.13	10.81	1.48
Teeth 22, 27	0.15	1.73	1.45	1.69	0.40
Tooth 6	1.74	2.58	7.34	9.93	1.18
Tooth 11	1.90	1.40	6.95	11.65	1.78
Tooth 22	0.13	1.83	1.42	1.84	0.31
Tooth 27	0.19	1.60	1.49	1.54	0.51
First bicuspids					
Teeth 5, 12, 21, 28	0.71	2.32	6.13	12.80	28.06
Teeth 5, 12	1.10	2.00	9.52	21.00	40.64
Teeth 21, 28	0.33	2.31	2.86	5.07	15.54
Tooth 5	0.64	2.24	10.69	19.94	39.22
Tooth 12	1.61	1.72	8.21	22.14	42.07
Tooth 21	0.31	2.95	2.88	6.20	15.30
Tooth 28	0.37	1.63	2.87	3.92	15.82

TABLE 2—*Continued*

TEETH	LINGUAL	BUCCAL	MESIAL	DISTAL	OCCLUSAL
Second bicuspids					
Teeth 4, 13, 20, 29	0.74	2.29	11.16	15.40	42.62
Teeth 4, 13	0.99	0.76	14.96	18.94	43.36
Teeth 20, 29	0.50	3.75	7.42	11.90	41.84
Tooth 4	0.85	0.49	15.85	19.17	41.21
Tooth 13	1.14	1.05	14.06	18.71	45.52
Tooth 20	0.49	3.86	8.67	13.03	41.57
Tooth 29	0.49	3.65	6.16	10.77	42.11
First molars					
Teeth 3, 14, 19, 30	18.77	26.64	14.26	6.02	114.63
Teeth 3, 14	31.56	5.13	15.19	5.95	123.35
Teeth 19, 30	3.13	53.92	13.34	6.23	100.21
Tooth 3	31.70	4.31	17.66	6.12	124.26
Tooth 14	31.44	5.97	12.69	5.77	122.44
Tooth 19	2.89	57.41	13.99	6.94	99.68
Tooth 30	3.38	50.26	12.66	5.49	100.81
Second molars					
Teeth 2, 15, 18, 31	6.24	18.45	4.10	2.90	103.02
Teeth 2, 15	11.63	9.95	3.49	4.20	106.31
Teeth 18, 31	1.46	26.26	4.68	1.74	98.85
Tooth 2	11.17	10.22	3.62	3.85	106.57
Tooth 15	12.09	9.67	3.36	4.53	106.08
Tooth 18	1.61	27.35	4.75	1.71	99.39
Tooth 31	1.32	25.12	4.61	1.77	98.30
Third molars					
Teeth 1, 16, 17, 32	1.28	12.39	1.56	1.33	65.46
Teeth 1, 16	1.89	9.78	1.23	1.90	64.72
Teeth 17, 32	0.83	14.51	1.82	0.93	66.12
Tooth 1	1.81	8.32	1.38	1.81	62.91
Tooth 16	1.98	11.12	1.11	1.98	66.47
Tooth 17	0.74	14.18	1.96	1.21	65.59
Tooth 32	0.93	14.90	1.67	0.60	66.67
Total	4.21	6.93	10.82	8.98	41.65
Upper teeth 1–16	7.33	3.46	16.17	13.09	45.66
Lower teeth 17–32	0.73	11.18	4.96	4.47	37.98

(4) To obtain the figure for "occlusal," the sum was taken of (a) occlusal, (b) all surfaces, (c) compound with occlusal, and (d) *twice* the figure for "two or more occlusal."

Our observations extended over a period of more than twelve years, and included the examination of at least eighteen thousand individuals of ages ranging from 16 to 70 years. The data collected during this period have been analyzed and systematized in various ways, with results of which those in *figs. 14–18* are only a few typical samples. In submitting these for your consideration, I might make an explanation regarding teeth reported as "missing." The number of cavities in such teeth, and their location on the teeth, are of course unknown. The same remark applies to crowned or very badly decayed teeth. We shall err on the side of conservatism if we ascribe to each surface of a missing tooth the number of cavities found, on an average, in that surface among the teeth of that particular kind that were not missing. An allowance was made in this way by means of a formula suggested by Dr. Lotka, of the Statistical Bureau of the Metropolitan Life Insurance Company. While this formula, as already stated, gives a conservative correction, it enables us to secure a better picture of the relative susceptibilities of the different tooth surfaces than if no correction were made for the missing teeth.

Our records are secured from an examination of many thousands of persons. These persons are not confined to one class. There are represented in these figures the dental conditions of plumbers, bricklayers, carpenters, electricians, clerks, commissary-department section heads, stenographers, executives, and professional men and women. This gives us a very good cross section of society as found today in New York City. It also includes many persons of different races. In all probability, if dental records were secured from other large cities, they would be found to approximate very closely the records secured in our Clinic.

ADDENDUM BY DR. LOTKA

The Statistical Bureau of the Metropolitan Life Insurance Company coöperates with the various departments of the Home Office in collecting data and subjecting them to statistical analysis. A great variety of problems comes to the Bureau, whose function it is to make a

scientific analysis of the facts, and, as occasion arises, to develop methods or to recommend procedures based on the data furnished. Among the questions that come to the department are problems relating to various aspects of hygiene, and, as in the present case, problems of dental hygiene. When Dr. Hyatt had carried his collection of observational data on dental examinations to a point where somewhat detailed statistical treatment became necessary, he entered into consultation with the Bureau and it fell to my share to engage in what has proved to be a very delightful coöperation with Dr. Hyatt, though my part in this work has, of course, been entirely subsidiary. Dr. Hyatt has presented to you the general account of the procedure followed in the dental investigation, and has also shown you some of the results presented in graphic form. There remains little more for me to say. Dr. Hyatt has asked me to speak to you on certain points that are brought out in *Table 3* and in *figs. 21* and *22*.

Figs. 21 and *22* have been prepared to show the relative susceptibility of the several tooth surfaces. As Dr. Hyatt has already intimated, correction was first applied for teeth missing or so badly decayed that it was not feasible definitely to count cavities on them. Each such "missing" tooth and each of its surfaces was assigned the average number of cavities found upon that surface in the corresponding defective teeth present in the mouth, upon which an actual count had been made. Since missing or badly decayed teeth would, from the nature of things, have more than the average number of cavities, this correction errs on the conservative side. After applying this correction for missing teeth, the proportion of cavities found on any particular surface, such as second molar occlusal, was expressed in per cent of the total cavities on all teeth (including those missing, credited, as explained, with their proper number of cavities). So, for example, the upper first molar occlusal surface had about 6.5 per cent of all cavities, and the lower about 5.2 per cent, giving, for the occlusal surfaces of the four first molars, a total of 11.7 per cent of *all* cavities. Now there are, in a full set of teeth, $32 \times 5 = 160$ surfaces. There are four first molar occlusal surfaces. If all surfaces of all teeth were equally susceptible, the four occlusal surfaces of the first molars would receive $\frac{4}{160} = \frac{1}{40}$ or 2.5 per cent of all cavities. They actually receive exactly almost five times this, namely, 11.7, say 12 per cent.

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

First part

	CENTRALS										LATERALS									
	Upper 8, 9					Lower 24, 25					Upper 7, 10					Lower 23, 26				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
Centrals																				
Upper 8, 9																				
Lingual	1.00	0.49	9.91	8.03	0.14	0.01	0.04	1.22	1.06	0.03	3.64	0.51	9.97	3.85	0.16	0.04	0.11	0.97	0.88	0.01
Buccal	2.05	1.00	20.35	16.48	0.29	0.02	0.08	2.50	2.18	0.06	7.48	1.05	20.47	7.91	0.32	0.08	0.22	1.98	1.80	0.03
Mesial	0.10	0.05	1.00	0.81	0.01	—	—	0.12	0.11	—	0.37	0.05	1.01	0.39	0.02	—	0.01	0.10	0.09	—
Distal	0.12	0.06	1.23	1.00	0.02	—	0.01	0.15	0.13	—	0.45	0.06	1.24	0.48	0.02	—	0.01	0.12	0.11	—
Occlusal	7.18	3.50	71.21	57.68	1.00	0.05	0.29	8.74	7.63	0.21	26.18	3.66	71.63	27.68	1.13	0.26	0.76	6.95	6.32	0.11
Lower 24, 25																				
Lingual	136.50	66.50	1353.00	1096.00	19.00	1.00	5.50	166.00	145.00	4.00	497.50	69.50	1361.00	526.00	21.50	5.00	14.50	132.00	120.00	2.00
Buccal	24.82	12.09	24.60	199.27	3.45	0.18	1.00	30.18	26.36	0.73	0.45	12.64	247.45	95.64	3.91	0.91	2.64	24.00	21.82	0.36
Mesial	0.82	0.40	8.15	6.60	0.11	0.01	0.03	1.00	0.87	0.02	3.00	0.42	8.20	3.17	0.13	0.03	0.09	0.80	0.72	0.01
Distal	0.94	0.46	9.33	7.56	0.13	0.01	0.04	1.14	1.00	0.03	3.43	0.48	9.39	3.63	0.15	0.03	0.10	0.91	0.83	0.01
Occlusal	34.13	16.63	338.25	274.00	4.75	0.25	1.38	41.50	36.25	1.00	124.38	17.38	340.25	131.50	5.38	1.25	3.63	33.00	30.00	0.50
Laterals																				
Upper 7, 10																				
Lingual	0.27	0.13	2.72	2.20	0.04	—	0.01	0.33	0.29	0.01	1.00	0.14	2.74	1.06	0.04	0.01	0.03	0.27	0.24	—
Buccal	1.96	0.96	19.47	15.77	0.27	0.01	0.08	2.39	2.09	0.06	7.16	1.00	19.58	7.57	0 31	0.07	0.21	1.90	1.73	0.03
Mesial	0.10	0.05	0.99	0.81	0.01	—	—	0.12	0.11	—	0.37	0.05	1.00	0.39	0 02	—	0.01	0.10	0.09	—
Distal	0.26	0.13	2.57	2.08	0.04	—	0.01	0.32	0.28	0.01	0.95	0.13	2.59	1.00	0.04	0 01	0.03	0.25	0.23	—
Occlusal	6.35	3.09	62.93	50.98	0.88	0.05	0.26	7.72	6.74	0.19	23.14	3.23	63.30	24.47	1.00	0.23	0.67	6.14	5.58	0.09
Lower 23, 26																				
Lingual	27.30	13.30	270.60	219.20	3.80	0.20	1.10	33.20	29.00	0.80	99.50	13.90	272.20	105.20	4.30	1.00	2.90	26.40	24.00	0.40
Buccal	9.41	4.59	93.31	75.59	1.31	0.07	0.38	11.45	10.00	0.28	34.31	4.79	93.86	36.28	1.48	0.34	1.00	9.10	8.28	0.14
Mesial	1.03	0.50	10.25	8.30	0.14	0.01	0.04	1.26	1.10	0.03	3.77	0.53	10.31	3.98	0.16	0.04	0.11	1.00	0.91	0.02
Distal	1.14	0.55	11.28	9.13	0.16	0.01	0.05	1.38	1.21	0.03	4.15	0.58	11.34	4.38	0.18	0.04	0.12	1.10	1.00	0.02
Occlusal	68.25	33.25	676.50	54.80	9.50	0.50	2.75	83.00	72.50	2.00	248.75	34.75	680.50	263.00	10.75	2.50	7.25	66.00	60.00	1.00
Cuspids																				
Upper 6, 11																				
Lingual	1.84	0.90	18.28	14.81	0.26	0.01	0.07	2.24	1.96	0.05	6.72	0.94	18.39	7.11	0.29	0.07	0.20	1.78	1.62	0.03
Buccal	1.71	0.83	16.91	13.70	0.24	0.01	0.07	2.08	1.81	0.05	6.22	0.87	17.01	6.58	0.27	0.06	0.18	1.65	1.50	0.03
Mesial	0.47	0.23	4.67	3.78	0.07	—	0.02	0.57	0.50	0.01	1.72	0.24	4.69	1.81	0.07	0.02	0.05	0.46	0.41	0.01
Distal	0.31	0.15	3.08	2.50	0.04	—	0.01	0.38	0.33	0.01	1.13	0.16	3.10	1.20	0.05	0.01	0.03	0.30	0.27	—
Occlusal	3.03	1.48	30.07	24.36	0.42	0.02	0.12	3.69	3.22	0.09	11.06	1.54	30.24	11.69	0.48	0.11	0.32	2.93	2.67	0.04
Lower 22, 27																				
Lingual	19.50	9.50	193.29	156.57	2.71	0.14	0.79	23.71	20.71	0.57	71.07	9.93	194.43	75.14	3.07	0.71	2.07	18.86	17.14	0.29
Buccal	1.73	0.84	17.13	13.87	0.24	0.01	0.07	2.10	1.84	0.05	6.30	0.88	17.23	6.66	0.27	0.06	0.18	1.67	1.52	0.03
Mesial	2.05	1.00	20.35	16.48	0.29	0.02	0.08	2.50	2.18	0.06	7.48	1.05	20.47	7.91	0.32	0.08	0.22	1.98	1.80	0.03
Distal	1.76	0.86	17.46	14.14	0.25	0.01	0.07	2.14	1.87	0.05	6.42	0.90	17.56	6.79	0.28	0.06	0.19	1.70	1.55	0.03
Occlusal	9.10	4.43	90.20	73.07	1.27	0.07	0.37	11.07	9.67	0.27	33.17	4.63	90.73	35.07	1.43	0.33	0.97	8.80	8.00	0.13
1st Bicuspids																				
Upper 5, 12																				
Lingual	5.46	2.66	54.12	43.84	0.76	0.04	0.22	6.64	5.80	0.16	19.90	2.78	54.44	21.04	0.86	0.20	0.58	5.28	4.80	0.08
Buccal	2.90	1.41	28.79	23.32	0.40	0.02	0.12	3.53	3.09	0.09	10.59	1.48	28.96	11.19	0.46	0.11	0.31	2.81	2.55	0.04
Mesial	0.59	0.29	5.88	4.77	0.08	—	0.02	0.72	0.63	0.02	2.16	0.30	5.92	2.29	0.09	0.02	0.06	0.57	0.52	0.01
Distal	0.27	0.13	2.66	2.15	0.04	—	0.01	0.33	0.28	0.01	0.98	0.14	2.67	1.03	0.04	0.01	0.03	0.26	0.24	—
Occlusal	0.13	0.06	1.26	1.02	0.02	—	0.01	0.15	0.14	—	0.46	0.06	1.27	0.49	0.02	—	0.01	0.12	0.11	—
Lower 21, 28																				
Lingual	15.17	7.39	150.33	121.78	2.11	0.11	0.61	18.44	16.11	0.44	55.28	7.72	151.22	58.44	2.39	0.56	1.61	14.67	13.33	0.22
Buccal	2.07	1.01	20.50	16.61	0.29	0.02	0.08	2.52	2.20	0.06	7.54	1.05	20.62	7.97	0.33	0.08	0.22	2.00	1.82	0.03
Mesial	1.66	0.81	16.50	13.37	0.23	0.01	0.07	2.02	1.77	0.05	6.07	0.85	16.60	6.41	0.26	0.06	0.18	1.61	1.46	0.02
Distal	0.93	0.46	9.27	7.51	0.13	0.01	0.04	1.14	0.99	0.03	3.41	0.48	9.32	3.60	0.15	0.03	0.10	0.90	0.82	0.01
Occlusal	0.28	0.14	2.80	2.27	0.04	—	0.01	0.34	0.30	0.01	1.03	0.14	2.82	1.09	0.04	0.01	0.03	0.27	0.25	—

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

Second part

	CUSPIDS										FIRST BICUSPIDS									
	Upper 6, 11					Lower 22, 27					Upper 5, 12					Lower 21, 28				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
Centrals																				
Upper 8, 9																				
Lingual	0.54	0.59	2.12	3.22	0.33	0.05	0.58	0.49	0.57	0.11	0.18	0.34	1.68	3.73	7.86	0.07	0.48	0.60	1.07	3.53
Buccal	1.11	1.20	4.36	6.60	0.68	0.11	1.19	1.00	1.17	0.23	0.38	0.71	3.46	7.66	16.14	0.14	0.99	1.23	2.20	7.26
Mesial	0.05	0.06	0.21	0.32	0.03	0.01	0.06	0.05	0.06	0.01	0.02	0.03	0.17	0.38	0.79	0.01	0.05	0.06	0.11	0.36
Distal	0.07	0.07	0.26	0.40	0.04	0.01	0.07	0.06	0.07	0.01	0.02	0.04	0.21	0.46	0.98	0.01	0.06	0.07	0.13	0.44
Occlusal	3.89	4.21	15.26	23.11	2.37	0.37	4.16	3.50	4.08	0.79	1.32	2.47	12.11	26.82	56.47	0.47	3.47	4.32	7.68	25.39
Lower 24, 25																				
Lingual	74.00	80.00	290.00	439.00	45.00	7.00	79.00	66.50	77.50	15.00	25.00	47.00	230.00	509.50	1073.00	9.00	66.00	82.00	146.00	482.50
Buccal	13.45	14.55	52.73	79.82	8.18	1.27	14.36	12.09	14.09	2.73	4.55	8.55	41.82	92.64	195.09	1.64	12.00	14.91	26.55	87.73
Mesial	0.45	0.48	1.75	2.64	0.27	0.04	0.48	0.40	0.47	0.09	0.15	0.28	1.39	3.07	6.46	0.05	0.40	0.49	0.88	2.91
Distal	0.51	0.55	2.00	3.03	0.31	0.05	0.54	0.46	0.53	0.10	0.17	0.32	1.59	3.51	7.40	0.06	0.46	0.57	1.01	3.33
Occlusal	18.50	20.00	72.50	109.75	11.25	1.75	19.75	16.63	19.38	3.75	6.25	11.75	57.50	127.38	268.25	2.25	16.50	20.50	36.50	120.63
Laterals																				
Upper 7, 10																				
Lingual	0.15	0.16	0.58	0.88	0.09	0.01	0.16	0.13	0.16	0.03	0.05	0.09	0.46	1.02	2.16	0.02	0.13	0.16	0.29	0.97
Buccal	1.06	1.15	4.17	6.32	0.65	0.10	1.14	0.96	1.12	0.22	0.36	0.68	3.31	7.33	15.44	0.13	0.95	1.18	2.10	6.94
Mesial	0.05	0.06	0.21	0.32	0.03	0.01	0.06	0.05	0.06	0.01	0.02	0.03	0.17	0.37	0.79	0.01	0.05	0.06	0.11	0.35
Distal	0.14	0.15	0.55	0.83	0.09	0.01	0.15	0.13	0.15	0.03	0.05	0.09	0.44	0.97	2.04	0.02	0.13	0.16	0.28	0.92
Occlusal	3.44	3.72	13.49	20.42	2.09	0.33	3.67	3.09	3.60	0.70	1.16	2.19	10.70	23.70	49.91	0.42	3.07	3.81	6.79	49.91
Lower 23, 26																				
Lingual	14.80	16.00	58.00	87.80	9.00	1.40	15.80	13.30	15.50	3.00	5.00	9.40	46.00	101.90	214.60	1.80	13.20	16.40	29.20	96.50
Buccal	5.10	5.52	20.00	30.28	3.10	0.48	5.45	4.59	5.34	1.03	1.72	3.24	15.86	35.14	74.00	0.62	4.55	5.66	10.07	33.28
Mesial	0.56	0.61	2.20	3.33	0.34	0.05	0.60	0.50	0.59	0.11	0.19	0.36	1.74	3.86	8.13	0.07	0.50	0.62	1.11	3.66
Distal	0.62	0.67	2.42	3.66	0.38	0.06	0.66	0.55	0.65	0.13	0.21	0.39	1.92	4.25	8.94	0.08	0.55	0.68	1.22	4.02
Occlusal	37.00	40.00	145.00	219.50	22.50	3.50	39.50	33.25	38.75	7.50	12.50	23.50	115.00	254.75	536.50	4.50	33.00	41.00	73.00	241.25
Cuspids																				
Upper 6, 11																				
Lingual	1.00	1.08	3.92	5.93	0.61	0.09	1.07	0.90	1.05	0.20	0.34	0.64	3.11	6.89	14.50	0.12	0.89	1.11	1.97	6.52
Buccal	0.93	1.00	3.63	5.49	0.56	0.09	0.99	0.83	0.97	0.19	0.31	0.59	2.88	6.37	13.41	0.11	0.83	1.03	1.83	6.03
Mesial	0.26	0.28	1.00	1.51	0.16	0.02	0.27	0.23	0.27	0.05	0.09	0.16	0.79	1.76	3.70	0.03	0.23	0.28	0.50	1.66
Distal	0.17	0.18	0.66	1.00	0.10	0.02	0.18	0.15	0.18	0.03	0.06	0.11	0.52	1.16	2.44	0.02	0.15	0.19	0.33	1.10
Occlusal	1.64	1.78	6.44	9.76	1.00	0.16	1.76	1.48	1.72	0.33	0.56	1.04	5.11	11.32	23.84	0.20	1.47	1.82	3.24	10.72
Lower 22, 27																				
Lingual	10.57	11.43	41.43	62.71	6.43	1.00	11.29	9.50	11.07	2.14	3.57	6.71	32.86	72.79	153.29	1.29	9.43	11.71	20.86	68.93
Buccal	0.94	1.01	3.67	5.56	0.57	0.09	1.00	0.84	0.98	0.19	0.32	0.59	2.91	6.45	13.58	0.11	0.84	1.04	1.85	6.11
Mesial	1.11	1.20	4.36	6.60	0.68	0.11	1.19	1.00	1.17	0.23	0.38	0.71	3.46	7.66	16.14	0.14	0.99	1.23	2.20	7.26
Distal	0.95	1.03	3.74	5.66	0.58	0.09	1.02	0.86	1.00	0.19	0.32	0.61	2.97	6.57	13.85	0.12	0.85	1.06	1.88	6.23
Occlusal	4.93	5.33	19.33	29.27	3.00	0.47	5.27	4.43	5.17	1.00	1.67	3.13	15.33	33.97	71.53	0.60	4.40	5.47	9.73	32.17
1st Bicuspids																				
Upper 5, 12																				
Lingual	2.96	3.20	11.60	17.56	1.80	0.28	3.16	2.66	3.10	0.60	1.00	1.88	9.20	20.38	42.92	0.36	2.64	3.28	5.84	19.30
Buccal	1.57	1.70	6.17	9.34	0.96	0.15	1.68	1.41	1.65	0.32	0.53	1.00	4.89	10.84	22.83	0.19	1.40	1.74	3.11	10.27
Mesial	0.32	0.35	1.26	1.91	0.20	0.03	0.34	0.29	0.34	0.07	0.11	0.20	1.00	2.22	4.67	0.04	0.29	0.36	0.63	2.10
Distal	0.15	0.16	0.57	0.86	0.09	0.01	0.16	0.13	0.15	0.03	0.05	0.09	0.45	1.00	2.11	0.02	0.13	0.16	0.29	0.95
Occlusal	0.07	0.07	0.27	0.41	0.04	0.01	0.07	0.06	0.07	0.01	0.02	0.04	0.21	0.47	1.00	0.01	0.06	0.08	0.14	0.45
Lower 21, 28																				
Lingual	8.22	8.89	32.22	48.78	5.00	0.78	8.78	7.39	8.61	1.67	2.78	5.22	25.56	56.61	119.22	1.00	7.33	9.11	16.22	53.61
Buccal	1.12	1.21	4.39	6.65	0.68	0.11	1.20	1.01	1.17	0.23	0.38	0.71	3.48	7.72	16.26	0.14	1.00	1.24	2.21	7.31
Mesial	0.90	0.98	3.54	5.35	0.55	0.09	0.96	0.81	0.95	0.18	0.30	0.57	2.80	6.21	13.09	0.11	0.80	1.00	1.78	5.88
Distal	0.51	0.55	1.99	3.01	0.31	0.05	0.54	0.46	0.53	0.10	0.17	0.32	1.58	3.49	7.35	0.06	0.45	0.56	1.00	3.30
Occlusal	0.15	0.17	0.60	0.91	0.09	0.01	0.16	0.14	0.16	0.03	0.05	0.10	0.48	1.06	2.22	0.02	0.14	0.17	0.30	1.00

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

Third part

	CENTRALS										LATERALS									
	Upper 8, 9					Lower 24, 25					Upper 7, 10					Lower 23, 26				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
2nd Bicuspids																				
Upper 4, 13																				
Lingual	0.58	0.28	5.76	4.66	0.08	—	0.02	0.71	0.62	0.02	2.12	0.30	5.79	2.24	0.09	0.02	0.06	0.56	0.51	0.01
Buccal	0.76	0.37	7.52	6.09	0.11	0.01	0.03	0.92	0.81	0.02	2.76	0.39	7.56	2.92	0.12	0.03	0.08	0.73	0.67	0.01
Mesial	0.38	0.18	3.72	3.02	0.05	—	0.02	0.46	0.26	0.01	1.37	0.19	3.74	1.45	0.06	0.01	0.04	0.36	0.33	0.01
Distal	0.30	0.14	2.94	2.38	0.04	—	0.01	0.36	0.31	0.01	1.08	0.15	2.96	1.14	0.05	0.01	0.03	0.29	0.26	—
Occlusal	0.11	0.05	1.10	0.89	0.02	—	—	0.13	0.12	—	0.40	0.06	1.10	0.43	0.02	—	0.01	0.11	0.10	—
Lower 20, 29																				
Lingual	13.65	6.65	135.30	109.60	1.90	0.10	0.55	16.60	14.50	0.40	49.75	6.95	136.10	52.60	2.15	0.50	1.45	13.20	12.00	0.20
Buccal	1.52	0.74	15.03	12.18	0.21	0.01	0.06	1.84	1.61	0.04	5.53	0.77	15.12	5.84	0.24	0.06	0.16	1.47	1.33	0.02
Mesial	0.76	0.37	7.52	6.09	0.11	0.01	0.03	0.92	0.81	0.02	2.77	0.39	7.56	2.92	0.12	0.03	0.08	0.74	0.67	0.01
Distal	0.47	0.23	4.67	3.78	0.07	—	0.02	0.57	0.50	0.01	1.72	0.24	4.69	1.81	0.07	0.02	0.05	0.46	0.41	0.01
Occlusal	0.10	0.05	1.02	0.83	0.01	—	—	0.12	0.11	—	0.37	0.05	1.02	0.40	0.02	—	0.01	0.10	0.09	—
1st Molars																				
Upper 3, 14																				
Lingual	0.17	0.08	1.67	1.35	0.02	—	0.01	0.20	0.18	—	0.61	0.09	1.68	0.65	0.03	0.01	0.02	0.16	0.15	—
Buccal	1.09	0.53	10.78	8.73	0.15	0.01	0.04	1.32	1.16	0.03	3.96	0.55	10.84	4.19	0.17	0.04	0.12	1.05	0.96	0.02
Mesial	0.35	0.17	3.50	2.83	0.05	—	0.01	0.43	0.37	0.01	1.29	0.18	3.52	1.36	0.06	0.01	0.04	0.34	0.31	0.01
Distal	0.93	0.45	9.20	7.46	0.13	0.01	0.04	1.13	0.99	0.03	3.38	0.47	9.26	3.58	0.15	0.03	0.10	0.90	0.82	0.01
Occlusal	0.07	0.03	0.65	0.53	0.01	—	—	0.08	0.07	—	0.24	0.03	0.65	0.25	0.01	—	0.01	0.06	0.06	—
Lower 19, 30																				
Lingual	1.78	0.87	17.69	14.33	0.25	0.01	0.07	2.17	1.90	0.05	6.50	0.91	17.79	6.88	0.28	0.07	0.19	1.73	1.57	0.03
Buccal	0.08	0.04	0.82	0.66	0.01	—	—	0.10	0.09	—	0.30	0.04	0.82	0.32	0.01	—	0.01	0.08	0.07	—
Mesial	0.35	0.17	3.43	2.78	0.05	—	0.01	0.42	0.37	0.01	1.26	0.18	3.45	1.33	0.05	0.01	0.04	0.33	0.30	0.01
Distal	0.79	0.38	7.82	6.34	0.11	0.01	0.03	0.96	0.84	0.02	2.88	0.40	7.87	3.04	0.12	0.03	0.08	0.76	0.69	0.01
Occlusal	0.05	0.02	0.47	0.38	0.01	—	—	0.06	0.05	—	0.17	0.02	0.48	0.18	0.01	—	0.01	0.05	0.04	—
2nd Molars																				
Upper 2, 15																				
Lingual	0.40	0.19	3.93	3.18	0.06	—	0.02	0.48	0.42	0.01	1.44	0.20	3.95	1.53	0.06	0.01	0.04	0.38	0.35	0.01
Buccal	0.46	0.23	4.60	3.73	0.06	—	0.02	0.56	0.49	0.01	1.69	0.24	4.63	1.79	0.07	0.02	0.05	0.45	0.41	0.01
Mesial	1.36	0.66	13.46	10.91	0.19	0.01	0.05	1.65	1.44	0.04	4.95	0.69	13.54	5.23	0.21	0.05	0.14	1.31	1.19	0.02
Distal	1.12	0.55	11.09	8.98	0.16	0.01	0.05	1.36	1.19	0.03	4.08	0.57	11.16	4.31	0.18	0.04	0.12	1.08	0.98	0.02
Occlusal	0.05	0.03	0.54	0.44	0.01	—	—	0.07	0.06	—	0.20	0.03	0.54	0.21	0.01	—	0.01	0.05	0.05	—
Lower 18, 31																				
Lingual	2.76	1.34	27.33	22.14	0.38	0.02	0.11	3.35	2.93	0.08	10.05	1.40	27.49	10.63	0.43	0.10	0.29	2.67	2.42	0.04
Buccal	0.14	0.07	1.42	1.15	0.02	—	0.01	0.17	0.15	—	0.52	0.07	1.43	0.55	0.02	0.01	0.02	0.14	0.13	—
Mesial	0.82	0.40	8.10	6.56	0.11	0.01	0.03	0.99	0.87	0.02	2.98	0.42	8.15	3.15	0.13	0.03	0.09	0.79	0.72	0.01
Distal	2.27	1.11	22.55	18.27	0.32	0.02	0.09	2.77	2.42	0.07	8.29	1.16	22.68	8.77	0.36	0.08	0.24	2.20	2.00	0.03
Occlusal	0.04	0.02	0.37	0.30	0.01	—	—	0.05	0.04	—	0.14	0.02	0.37	0.14	0.01	—	—	0.04	0.03	—
3rd Molars																				
Upper 1, 16																				
Lingual	1.99	0.97	19.75	16.00	0.28	0.01	0.08	2.42	2.12	0.06	7.26	1.01	19.87	7.68	0.31	0.07	0.21	1.93	1.75	0.03
Buccal	0.39	0.19	3.83	3.10	0.05	—	0.02	0.47	0.41	0.01	1.41	0.20	3.85	1.49	0.06	0.01	0.04	0.37	0.34	0.01
Mesial	3.07	1.49	30.40	24.63	0.43	0.02	0.12	3.73	3.26	0.09	11.18	1.56	30.58	11.82	0.48	0.11	0.33	2.97	2.70	0.04
Distal	1.99	0.97	19.75	16.00	0.28	0.01	0.08	2.42	2.12	0.06	7.26	1.01	19.87	7.68	0.31	0.07	0.21	1.93	1.75	0.03
Occlusal	0.05	0.03	0.52	0.42	0.01	—	—	0.06	0.06	—	0.19	0.03	0.52	0.20	0.01	—	0.01	0.05	0.05	—
Lower 17, 32																				
Lingual	4.48	2.18	44.36	35.93	0.62	0.03	0.18	5.44	4.75	0.13	16.31	2.28	44.62	17.25	0.70	0.16	0.48	4.33	3.93	0.07
Buccal	0.23	0.11	2.30	1.86	0.03	—	0.01	0.28	0.25	0.01	0.85	0.12	2.31	0.89	0.89	0.01	0.02	0.22	0.20	—
Mesial	1.92	0.94	19.06	15.44	0.27	0.01	0.08	2.34	2.04	0.06	7.01	0.98	19.17	7.41	0.30	0.07	0.20	1.86	1.69	0.03
Distal	3.96	1.93	39.22	31.77	0.55	0.03	0.16	4.81	4.20	0.12	14.42	2.01	39.45	15.25	0.62	0.14	0.42	3.83	3.48	0.06
Occlusal	0.05	0.02	0.50	0.41	0.01	—	—	0.06	0.05	—	0.18	0.03	0.51	0.20	0.01	—	0.01	0.05	0.04	—

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

Fourth part

	CUSPIDS										FIRST BICUSPIDS									
	Upper 6, 11					Lower 22, 27					Upper 5, 12					Lower 21, 28				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
2nd Bicuspids																				
Upper 4, 13																				
Lingual	0.31	0.34	1.23	1.87	0.19	0.03	0.34	0.28	0.33	0.06	0.11	0.20	0.98	2.17	4.57	0.04	0.28	0.35	0.62	2.05
Buccal	0.41	0.44	1.61	2.44	0.25	0.04	0.44	0.37	0.43	0.83	0.14	0.26	1.28	2.83	5.96	0.05	0.37	0.46	0.81	2.68
Mesial	0.20	0.22	0.80	1.21	0.12	0.02	0.22	0.18	0.21	0.04	0.07	0.13	0.63	1.40	2.95	0.02	0.18	0.23	0.40	1.33
Distal	0.16	0.17	0.63	0.95	0.10	0.02	0.17	0.14	0.17	0.03	0.05	0.10	0.50	1.11	2.33	0.02	0.14	0.18	0.32	1.05
Occlusal	0.06	0.06	0.24	0.36	0.04	0.01	0.06	0.05	0.06	0.01	0.02	0.04	0.19	0.41	0.87	0.01	0.05	0.07	0.12	0.39
Lower 20, 29																				
Lingual	7.40	8.00	29.00	43.90	4.50	0.70	7.90	6.65	7.75	1.50	2.50	4.70	23.00	50.95	107.30	0.90	6.60	8.20	14.60	48.25
Buccal	0.82	0.89	3.22	4.88	0.50	0.08	0.88	0.74	0.86	0.17	0.28	0.52	2.56	5.66	11.92	0.10	0.73	0.91	1.62	5.36
Mesial	0.41	0.45	1.61	2.44	0.25	0.04	0.44	0.37	0.43	0.09	0.14	0.26	1.28	2.83	5.96	0.05	0.37	0.46	0.81	2.68
Distal	0.26	0.28	1.00	1.51	0.16	0.02	0.27	0.23	0.27	0.05	0.09	0.16	0.79	1.76	3.70	0.03	0.23	0.28	0.50	1.66
Occlusal	0.06	0.06	0.22	0.33	0.03	0.01	0.06	0.05	0.06	0.01	0.02	0.04	0.17	0.38	0.81	0.01	0.05	0.06	0.11	0.36
1st Molars																				
Upper 3, 14																				
Lingual	0.09	0.10	0.36	0.54	0.06	0.01	0.10	0.08	0.10	0.02	0.03	0.06	0.28	0.63	1.32	0.01	0.08	0.10	0.18	0.59
Buccal	0.59	0.64	2.31	3.50	0.36	0.06	0.63	0.53	0.62	0.12	0.20	0.37	1.83	4.06	8.55	0.07	0.53	0.65	1.16	3.84
Mesial	0.19	0.21	0.75	1.13	0.12	0.02	0.20	0.17	0.20	0.04	0.06	0.12	0.59	1.32	2.77	0.02	0.17	0.21	0.38	1.25
Distal	0.50	0.54	1.97	2.99	0.31	0.05	0.54	0.45	0.53	0.10	0.17	0.32	1.56	3.47	7.30	0.06	0.45	0.56	0.99	3.28
Occlusal	0.04	0.04	0.14	0.21	0.02	—	0.04	0.03	0.04	—	0.01	0.02	0.11	0.24	0.52	—	0.03	0.04	0.07	0.23
Lower 19, 30																				
Lingual	0.97	1.05	3.79	5.74	0.59	0.09	1.03	0.87	1.01	0.20	0.33	0.61	3.01	6.66	14.03	0.12	0.86	1.07	1.91	6.31
Buccal	0.04	0.05	0.17	0.26	0.03	—	0.05	0.04	0.05	0.01	0.02	0.03	0.14	0.31	0.65	0.01	0.04	0.05	0.09	0.29
Mesial	0.19	0.20	0.74	1.11	0.11	0.02	0.20	0.17	0.20	0.04	0.06	0.12	0.58	1.29	2.72	0.02	0.17	0.21	0.37	1.22
Distal	0.43	0.46	1.68	2.54	0.26	0.04	0.46	0.38	0.45	0.09	0.14	0.27	1.33	2.95	6.20	0.05	0.38	0.47	0.84	2.79
Occlusal	0.03	0.03	0.10	0.15	0.02	—	0.03	0.02	0.03	0.01	0.01	0.02	0.08	0.18	0.38	—	0.02	0.03	0.05	0.17
2nd Molars																				
Upper 2, 15																				
Lingual	0.21	0.23	0.84	1.27	0.13	0.02	0.23	0.19	0.22	0.04	0.07	0.14	0.67	1.48	3.11	0.03	0.19	0.24	0.42	1.40
Buccal	0.25	0.27	0.99	1.49	0.15	0.02	0.27	0.23	0.26	0.05	0.09	0.16	0.78	1.73	3.65	0.03	0.22	0.28	0.50	1.64
Mesial	0.74	0.80	2.89	4.37	0.45	0.07	0.79	0.66	0.77	0.15	0.25	0.47	2.29	5.07	10.68	0.09	0.66	0.82	1.45	4.80
Distal	0.61	0.66	2.38	3.60	0.37	0.06	0.65	0.55	0.64	0.12	0.20	0.39	1.89	4.18	8.80	0.07	0.54	0.67	1.20	3.95
Occlusal	0.03	0.03	0.12	0.18	0.02	—	0.03	0.03	0.03	0.01	0.01	0.02	0.09	0.20	0.43	—	0.03	0.03	0.06	0.19
Lower 18, 31																				
Lingual	1.49	1.62	5.86	8.87	0.91	0.14	1.60	1.34	1.57	0.30	0.51	0.95	4.65	10.29	21.68	0.18	1.33	1.66	2.95	9.75
Buccal	0.08	0.08	0.30	0.46	0.05	0.01	0.08	0.07	0.08	0.02	0.03	0.05	0.24	0.53	1.12	0.01	0.07	0.09	0.15	0.51
Mesial	0.44	0.48	1.74	2.63	0.27	0.04	0.47	0.40	0.46	0.09	0.15	0.28	1.38	3.05	6.43	0.05	0.40	0.49	0.87	2.89
Distal	1.23	1.33	4.83	7.32	0.75	0.12	1.32	1.11	1.29	0.25	0.42	0.78	3.83	8.49	17.88	0.15	1.10	1.37	2.43	8.04
Occlusal	0.02	0.02	0.08	0.12	0.01	—	0.02	0.02	0.02	—	0.01	0.01	0.06	0.14	0.29	—	0.02	0.02	0.04	0.13
3rd Molars																				
Upper 1, 16																				
Lingual	1.08	1.17	4.23	6.41	0.66	0.10	1.15	0.97	1.13	0.22	0.36	0.69	3.36	7.44	15.66	0.13	0.96	1.20	2.13	7.04
Buccal	0.21	0.23	0.82	1.24	0.13	0.02	0.22	0.19	0.22	0.04	0.07	0.13	0.65	1.44	3.04	0.03	0.19	0.23	0.41	1.36
Mesial	1.66	1.80	6.52	9.87	1.01	0.16	1.78	1.49	1.74	0.34	0.56	1.06	5.17	11.45	24.11	0.20	1.48	1.84	3.28	10.84
Distal	1.08	1.17	4.23	6.41	0.66	0.10	1.15	0.97	1.13	0.22	0.36	0.69	3.36	7.44	15.66	0.13	0.96	1.20	2.13	7.04
Occlusal	0.03	0.03	0.11	0.17	0.02	—	0.03	0.03	0.03	0.01	0.01	0.02	0.09	0.20	0.41	—	0.03	0.03	0.06	0.19
Lower 17, 32																				
Lingual	2.43	2.62	9.51	14.39	1.48	0.23	2.59	2.18	2.54	0.49	0.82	1.54	7.54	16.70	35.18	0.30	2.16	2.69	4.79	15.82
Buccal	0.13	0.14	0.49	0.75	0.08	0.01	0.13	0.11	0.13	0.03	0.04	0.08	0.39	0.87	1.82	0.02	0.11	0.14	0.25	0.82
Mesial	1.04	1.13	4.08	6.18	0.63	0.10	1.11	0.94	1.09	0.21	0.35	0.66	3.24	7.18	15.11	0.13	0.93	1.15	2.06	6.80
Distal	2.14	2.32	8.41	12.72	1.30	0.20	2.29	1.93	2.25	0.43	0.72	1.36	6.67	14.77	31.10	0.26	1.91	2.38	4.23	13.99
Occlusal	0.03	0.03	0.11	0.16	0.02	—	0.03	0.02	0.03	0.01	0.01	0.02	0.09	0.19	0.40	—	0.02	0.03	0.05	0.18

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

Fifth part

	SECOND BICUSPIDS										FIRST MOLARS									
	Upper 4, 13					Lower 20, 29					Upper 3, 14					Lower 19, 30				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
2nd Bicuspids																				
Upper 4, 13																				
Lingual	1.00	0.77	15.47	19.60	52.51	0.43	3.83	7.66	12.34	56.51	34.57	5.34	16.47	6.26	88.51	3.26	70.53	16.79	7.36	121.68
Buccal	1.31	1.00	20.19	25.58	68.56	0.56	5.00	10.00	16.11	73.78	45.14	6.97	21.50	8.17	115.56	4.25	92.08	21.92	9.61	158.86
Mesial	0.06	0.05	1.00	1.27	3.39	0.03	0.25	0.50	0.80	3.65	2.24	0.35	1.06	0.40	5.72	0.21	4.56	1.09	0.48	7.87
Distal	0.05	0.04	0.79	1.00	2.68	0.02	0.20	0.28	0.63	2.88	1.76	0.27	0.84	0.32	4.52	0.17	3.60	0.86	0.38	6.21
Occlusal	0.02	0.01	0.29	0.37	1.00	0.01	0.07	0.15	0.24	1.08	0.66	0.10	0.31	0.12	1.69	0.06	1.34	0.32	0.14	2.32
Lower 20, 29																				
Lingual	2.35	1.80	36.35	46.05	123.40	1.00	9.00	18.00	29.00	132.80	81.25	12.55	38.70	14.70	208.00	7.65	165.75	39.45	17.30	285.95
Buccal	0.26	0.20	4.04	5.12	13.71	0.11	1.00	2.00	3.22	14.76	9.03	1.39	4.30	1.63	23.11	0.85	18.42	4.38	1.92	31.77
Mesial	0.13	0.10	2.02	2.56	6.86	0.06	0.50	1.00	1.61	7.38	4.51	0.70	2.15	0.82	11.56	0.43	9.21	2.19	0.96	15.89
Distal	0.08	0.06	1.25	1.59	4.26	0.03	0.31	0.62	1.00	4.58	2.80	0.43	1.33	0.51	7.17	0.26	5.72	1.36	0.60	9.86
Occlusal	0.02	0.01	0.27	0.35	0.93	0.01	0.07	0.14	0.22	1.00	0.61	0.09	0.29	0.11	1.57	0.06	1.25	0.30	0.13	2.15
1st Molars																				
Upper 3, 14																				
Lingual	0.03	0.02	0.45	0.57	1.52	0.01	0.11	0.22	0.36	1.63	1.00	0.15	0.48	0.18	2.56	0.09	2.04	0.49	0.21	3.52
Buccal	0.19	0.14	2.90	3.67	9.83	0.08	0.72	1.43	2.31	10.58	6.47	1.00	3.08	1.17	16.57	0.61	13.21	3.14	1.38	22.78
Mesial	0.06	0.05	0.94	1.19	3.19	0.03	0.23	0.47	0.75	3.43	2.10	0.32	1.00	0.37	5.37	0.20	4.28	1.02	0.45	7.39
Distal	0.16	0.12	2.47	3.13	8.39	0.07	0.61	1.22	1.97	9.03	5.53	0.85	2.63	1.00	14.15	0.52	11.28	2 68	1.18	19.45
Occlusal	0.11	0.09	1.75	2.21	5.93	0.05	0.43	0.87	1.39	6.38	3.91	0.60	1.86	0.71	1.00	0.37	7.97	1.90	0.83	13.75
Lower 19, 30																				
Lingual	0.31	0.24	4.75	6.02	16.13	0.13	1.18	2.35	3.79	17.36	10.62	1.64	5.06	1.92	27.19	1.00	21.67	5.16	2.26	37.38
Buccal	0.01	0.01	0.22	0.28	0.74	0.01	0.05	0.11	0.17	0.80	0.49	0.08	0.23	0.09	1.25	0.05	1.00	0.24	0.10	1.73
Mesial	0.06	0.05	0.92	1.17	3.13	0.03	0.23	0.46	0.74	3.37	2.06	0.32	0.98	0.37	5.27	0.19	4.20	1.00	0.44	7.25
Distal	0.14	0.10	2.10	2.66	7.13	0.06	0.52	1.04	1.68	7.68	4.70	0.73	2.24	0.85	12.02	0.44	9.58	2.28	1.00	16.53
Occlusal	0.08	0.06	1.27	1.61	4.32	0.03	0.31	0.63	1.01	4.64	2.84	0.44	1.35	0.51	7.27	0.27	5.80	1.38	0.61	1.00
2nd Molars																				
Upper 2, 15																				
Lingual	0.07	0.05	1.06	1.34	3.58	0.03	0.26	0.52	0.84	3.85	2.36	0.36	1.12	0.43	6.04	0.22	4.81	1.15	0.50	8.30
Buccal	0.08	0.06	1.24	1.57	4.20	0.03	0.31	0.61	0.99	4.52	2.76	0.43	1.32	0.50	7.07	0.26	5.64	1.34	0.59	9.73
Mesial	0.23	0.18	3.62	4.63	12.27	0.10	0.90	1.79	2.89	13.21	8.08	1.25	3.85	1.46	20.70	0.76	16.49	3.93	1.72	28.45
Distal	0.19	0.15	2.98	3.77	10.11	0.08	0.74	1.48	2.38	10.89	6.66	1.03	3.17	1.20	17.05	0.63	13.59	3.23	1.42	23.44
Occlusal	0.01	0.01	0.15	0.18	0.49	—	0.04	0.07	0.12	0.53	0.33	0.05	0.15	0.06	0.83	0.03	0.66	0.16	0.07	1.14
Lower 18, 31																				
Lingual	0.47	0.36	7.34	9.30	24.93	0.20	1.82	3.43	5.25	26.83	16.41	2.54	7.82	2.97	42.02	1.55	33.48	7.97	3.49	57.76
Buccal	0.25	0.19	3.81	4.83	12.94	0.10	0.94	1.89	3.04	13.92	8.52	1.32	4.06	1.54	21.80	0.80	17.37	4.14	1.81	29.97
Mesial	0.14	0.11	2.18	2.76	7.39	0.06	0.54	1.08	1.74	7.95	4.87	0.75	2.32	0.88	12.46	0.46	9.93	2.36	1.04	17.12
Distal	0.39	0.30	6.06	7.67	20.57	0.17	1.50	3.00	4.83	22.13	13.54	2.09	6.45	2.45	34.67	1.27	27.62	6.57	2.88	47.66
Occlusal	0.01	—	0.10	0.13	0.34	—	0.02	0.05	0.08	0.36	0.22	0.03	0.11	0.04	0.57	0.02	0.45	0.11	0.05	0.78
3rd Molars																				
Upper 1, 16																				
Lingual	0.34	0.26	5.31	6.72	18.01	0.15	1.31	2.63	4.23	19.39	11.86	1.83	5.65	2.15	30.36	1.12	24.20	5.76	2.53	41.74
Buccal	0.07	0.05	1.03	1.30	3.49	0.03	0.25	0.51	0.82	3.76	2.30	0.36	1.09	0.42	5.88	0.22	4.69	1.12	0.49	8.09
Mesial	0.53	0.40	8.17	10.35	27.73	0.22	2.02	4.04	6.52	29.84	18.26	2.82	8.70	3.30	46.74	1.72	37.25	8.87	3.89	64.26
Distal	0.34	0.26	5.31	6.72	18.01	0.15	1.31	2.63	4.23	19.39	11.86	1.83	5.65	2.15	30.36	1.12	24.20	5.76	2.53	41.74
Occlusal	0.01	0.01	0.14	0.18	0.47	—	0.03	0.07	0.11	0.51	0.31	0.05	0.15	0.06	0.80	0.03	0.64	0.15	0.07	1.10
Lower 17, 32																				
Lingual	0.77	0.59	11.92	15.10	40.46	0.33	2.95	5.90	9.51	43.54	26.64	4.11	12.69	4.82	68.20	2.51	54.34	12.93	5.67	93.75
Buccal	0.04	0.03	0.62	0.78	2.10	0.02	0.15	0.31	0.49	2.26	1.38	0.21	0.66	0.25	3.53	0.13	2.82	0.67	0.29	4.86
Mesial	0.33	0.25	5.12	6.49	17.38	0.14	1.27	2.54	4.08	18.70	11.44	1.77	5.45	2.07	29.30	1.08	23.35	5.56	2.44	40.27
Distal	0.68	0.52	10.54	13.35	35.77	0.29	2.61	5.22	8.41	38.49	23.55	3.64	11.22	4.26	60.29	2.22	48.04	11.44	5.01	82.88
Occlusal	0.01	0.01	0.13	0.17	0.46	—	0.03	0.07	0.11	0.49	0.30	0.05	0.14	0.05	0.77	0.03	0.62	0.15	0.06	1.06

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

Sixth part

	SECOND MOLARS										THIRD MOLARS									
	Upper 2, 15					Lower 18. 31					Upper 1, 16					Lower 17, 32				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
2nd Bicuspids																				
Upper 4, 13																				
Lingual	14.66	12.51	4.28	5.19	106.36	2.11	40.60	7.11	2.55	155.15	2.91	15.04	1.89	2.91	110.55	1.30	25.04	3.02	1.47	114.68
Buccal	19.14	16.33	5.58	6.78	138.86	2.75	53.00	9.28	3.33	202.56	3.81	19.64	2.47	3.81	144.33	1.69	32.69	3.94	1.92	149.72
Mesial	0.95	0.81	0.28	0.34	6.88	0.14	2.62	0.46	0.17	10.03	0.19	0.97	0.12	0.19	7.15	0.08	1.62	0.20	0.09	7.41
Distal	0.75	0.64	0.22	0.26	5.43	0.11	2.07	0.36	0.13	7.92	0.15	0.77	0.10	0.15	5.64	0.07	1.28	0.15	0.07	5.85
Occlusal	0.28	0.24	0.08	0.10	2.03	0.04	0.77	0.14	0.05	2.95	0.06	0.29	0.04	0.06	2.11	0.02	0.48	0.06	0.03	2.81
Lower 20, 29																				
Lingual	34.45	29.40	10.05	12.20	249.95	4.95	95.40	16.70	6.00	364.60	6.85	35.35	4.45	6.85	259.80	3.05	58.85	7.10	3.45	269.50
Buccal	3.83	3.27	1.12	1.36	27.77	0.55	10.60	1.86	0.67	40.51	0.76	3.93	0.49	0.76	28.87	0.34	6.54	0.79	0.38	29.94
Mesial	1.91	1.63	0.56	0.68	13.89	0.28	5.30	0.93	0.33	20.26	0.38	1.96	0.25	0.38	14.43	0.17	3.27	0.39	0.19	14.97
Distal	1.19	1.01	0.35	0.42	8.62	0.17	3.29	0.58	0.21	12.57	0.24	1.22	0.15	0.24	8.96	0.11	2.03	0.24	0.12	9.29
Occlusal	0.26	0.22	0.08	0.09	1.88	0.04	0.72	0.13	0.05	2.75	0.05	0.27	0.03	0.05	1.96	0.02	0.44	0.05	0.03	2.03
1st Molars																				
Upper 3, 14																				
Lingual	0.42	0.36	0.12	0.15	3.08	0.06	1.17	0.21	0.07	4.49	0.08	0.44	0.05	0.08	3.20	0.04	0.72	0.09	0.04	3.32
Buccal	2.75	2.34	0.80	0.97	19.92	0.39	7.60	1.33	0.48	29.05	0.55	2.82	0.35	0.55	20.70	0.24	4.69	0.57	0.27	21.47
Mesial	0.89	0.76	0.26	0.32	6.46	0.13	2.47	0.43	0.16	9.42	0.18	0.91	0.11	0.18	6.71	0.08	1.52	0.18	0.09	6.96
Distal	2.34	2.00	0.68	0.83	17.00	0.34	6.49	1.14	0.41	24.80	0.47	2.40	0.30	0.47	17.67	0.21	4.00	0.48	0.23	18.33
Occlusal	1.66	1.41	0.48	0.59	12.02	0.24	4.59	0.80	0.29	17.53	0.33	1.70	0.21	0.33	12.49	0.15	2.83	0.34	0.17	12.96
Lower 19, 30																				
Lingual	4.50	3.84	1.31	1.59	32.67	0.65	12.47	2.18	0.78	47.66	0.90	4.62	0.58	0.90	33.96	0.40	7.69	0.93	0.45	35.23
Buccal	0.21	0.18	0.06	0.07	1.51	0.03	0.58	0.10	0.04	2.20	0.04	0.21	0.03	0.04	1.57	0.02	0.36	0.04	0.02	1.63
Mesial	0.87	0.75	0.25	0.31	6.34	0.13	2.42	0.42	0.15	9.24	0.17	0.90	0.11	0.17	6.59	0.08	1.49	0.18	0.09	6.83
Distal	1.99	1.70	0.58	0.71	14.45	0.29	5.51	0.97	0.35	21.08	0.40	2.04	0.26	0.40	15.02	0.18	3.40	0.41	0.20	15.58
Occlusal	1.20	1.03	0.35	0.43	8.74	0.17	3.34	0.58	0.21	12.75	0.24	1.24	0.16	0.24	9.09	0.11	2.06	0.25	0.12	9.42
2nd Molars																				
Upper 2, 15																				
Lingual	1.00	0.85	0.29	0.35	7.26	0.14	2.77	0.48	0.17	10.58	0.20	1.03	0.13	0.20	7.54	0.09	1.71	0.21	0.10	7.82
Buccal	1.17	1.00	0.34	0.41	8.50	0.17	3.24	0.57	0.20	12.40	0.23	1.20	0.15	0.23	8.84	0.10	2.00	0.24	0.12	9.17
Mesial	3.43	2.93	1.00	1.21	24.87	0.49	9.49	1.66	0.60	36.28	0.68	3.52	0.44	0.68	25.85	0.30	5.86	0.71	0.34	26.82
Distal	2.82	2.41	0.82	1.00	20.49	0.41	7.82	1.37	0.49	29.89	0.56	2.90	0.36	0.56	21.30	0.25	4.82	0.58	0.28	22.09
Occlusal	0.14	0.12	0.04	0.05	1.00	0.02	0.38	0.07	0.02	1.46	0.03	0.14	0.02	0.03	1.04	0.01	0.24	0.03	0.01	1.08
Lower 18, 31																				
Lingual	6.96	5.94	2.03	2.46	50.49	1.00	19.27	3.37	1.21	73.65	1.38	7.14	0.90	1.38	52.48	0.62	11.89	1.43	0.70	54.44
Buccal	3.61	3.08	1.05	1.28	26.20	0.52	1.00	1.75	0.63	38.22	0.72	3.71	0.47	0.72	27.23	0.32	6.17	0.74	0.36	28.25
Mesial	2.06	1.76	0.60	0.73	14.97	0.30	5.71	1.00	0.36	21.83	0.41	2.12	0.27	0.41	15.56	0.18	3.52	0.43	0.21	16.14
Distal	5.74	4.90	1.67	2.03	41.66	0.82	15.90	2.78	1.00	60.77	1.14	5.89	0.74	1.14	43.30	0.51	9.81	1.18	0.57	44.92
Occlusal	0.09	0.08	0.03	0.03	0.69	0.01	0.26	0.05	0.02	1.00	0.02	0.10	0.01	0.02	0.71	0.01	0.16	0.02	0.01	0.74
3rd Molars																				
Upper 1, 16																				
Lingual	5.03	4.29	1.47	1.78	36.49	0.72	13.93	2.44	0.88	53.23	1.00	5.16	0.65	1.00	37.93	0.45	8.59	1.04	0.50	39.34
Buccal	0.97	0.83	0.28	0.35	7.07	0.14	2.70	0.47	0.17	10.31	0.19	1.00	0.13	0.19	7.35	0.09	1.66	0.20	0.10	7.62
Mesial	7.74	6.61	2.26	2.74	56.17	1.11	21.44	3.75	1.35	81.93	1.54	7.94	1.00	1.54	58.38	0.69	13.22	1.60	0.78	60.56
Distal	5.03	4.29	1.47	1.78	36.49	0.72	13.93	2.44	0.88	53.23	1.00	5.16	0.65	1.00	37.93	0.45	8.59	1.04	0.50	39.34
Occlusal	0.13	0.11	0.04	0.05	0.96	0.02	0.37	0.06	0.02	1.40	0.03	0.14	0.02	0.03	1.00	0.01	0.23	0.03	0.01	1.04
Lower 17, 32																				
Lingual	11.30	9.64	3.30	4.00	81.95	1.62	31.28	5.48	1.97	119.54	2.25	11.59	1.46	2.25	85.18	1.00	19.30	2.33	1.13	88.36
Buccal	0.59	0.50	0.17	0.21	4.25	0.08	1.62	0.28	0.10	6.20	0.12	0.60	0.08	0.12	4.41	0.05	1.00	0.12	0.06	4.58
Mesial	4.85	4.14	1.42	1.72	35.20	0.70	13.44	2.35	0.85	51.35	0.96	4.98	0.63	0.96	36.59	0.43	8.29	1.00	0.49	37.96
Distal	9.99	8.52	2.91	3.54	72.45	1.44	27.65	4.84	1.74	5.68	1.99	10.25	1.29	1.99	75.30	0.88	17.06	2.06	1.00	78.12
Occlusal	0.13	0.11	0.04	0.05	0.93	0.02	0.35	0.06	0.02	1.35	0.03	0.13	0.02	0.03	0 96	0.01	0.22	0.03	0.01	1.00

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

Seventh part

	SECOND BICUSPIDS										FIRST MOLARS									
	Upper 4, 13					Lower 20, 29					Upper 3, 14					Lower 19, 30				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
Centrals																				
Upper 8, 9																				
Lingual	0.17	0.13	2.66	3.37	9.04	0.07	0.66	1.32	2.12	9.73	5.95	0.92	2.84	1.08	15.24	0.56	12.14	2.89	1.27	20.95
Buccal	0.35	0.27	5.47	6.92	18.56	0.15	1.35	2.71	4.36	19.97	12.22	1.89	5.82	2.21	31.28	1.15	24.92	5.93	2.60	50.52
Mesial	0.02	0.01	0.27	0.34	0.91	0.01	0.07	0.13	0.21	0.98	0.60	0.09	0.29	0.11	1.54	0.06	1.23	0.25	0.13	2.15
Distal	0.02	0.02	0.33	0.42	1.13	0.01	0.08	0.16	0.26	1.21	0.74	0.11	0.35	0.13	1.90	0.07	1.51	0.36	0.16	2.61
Occlusal	1.24	0.95	19.13	24.24	64.95	0.53	4.74	9.47	15.26	69.89	42.76	6.61	20.37	7.74	109.47	4.03	87.24	20.76	9.11	150.50
Lower 24, 25																				
Lingual	23.50	18.00	363.50	460.50	1234.00	10.00	90.00	180.00	290.00	1328.00	812.50	125.50	387.00	147.00	2080.00	76.50	1657.50	394.50	173.00	2859.50
Buccal	4.27	3.27	66.09	83.73	224.36	1.82	16.36	32.73	52.73	241.45	147.73	22.82	70.36	26.73	378.18	13.91	301.36	71.73	31.45	519.91
Mesial	0.14	0.11	2.19	2.77	7.43	0.06	0.54	1.08	1.75	8.00	4.89	0.76	2.33	0.89	12.53	0.46	9.98	2.38	1.04	17.23
Distal	0.16	0.12	2.51	3.18	8.51	0.07	0.62	1.24	2.00	9.16	5.60	0.87	2.67	1.01	14.34	0.53	11.43	2.72	1.19	19.72
Occlusal	5.88	4.50	90.88	115.13	308.50	2.50	22.50	45.00	72.50	332.00	203.13	31.38	96.75	36.75	520.00	19.13	414.38	98.63	43.25	714.88
Laterals																				
Upper 7, 10																				
Lingual	0.05	0.04	0.73	0.93	2.48	0.02	0.18	0.36	0.58	2.67	1.63	0.25	0.78	0.30	4.18	0.15	3.33	0.79	0.35	5.75
Buccal	0.34	0.26	5.23	6.63	17.76	0.14	1.29	2.59	4.17	19.11	11.69	1.81	5.57	2.12	29.93	1.10	23.85	5.68	2.49	41.14
Mesial	0.02	0.01	0.27	0.34	0.91	0.01	0.07	0.13	0.21	0.98	0.60	0.09	0.28	0.11	1.53	0.06	1.22	0.29	0.13	2.10
Distal	0.04	0.03	0.69	0.88	2.35	0.02	0.17	0.34	0.55	2.52	1.54	0.24	0.74	0.28	3.95	0.15	3.15	0.75	0.33	5.44
Occlusal	1.09	0.84	16.91	21.42	57.40	0.47	4.19	8.37	13.49	61.77	37.79	5.84	1.80	6.84	96.74	3.56	77.09	18.35	8.05	133.00
Lower 23, 26																				
Lingual	4.70	3.60	72.70	92.10	246.80	2.00	18.00	36.00	58.00	265.60	162.50	25.10	77.40	29.40	416.00	15.30	331.50	78.90	34.60	571.90
Buccal	1.62	1.24	25.07	31.76	85.10	0.69	6.21	12.41	20.00	91.59	56.03	8.66	26.69	10.14	143.45	5.28	114.31	27.21	11.93	197.21
Mesial	0.18	0.14	2.75	3.49	9.35	0.08	0.68	1.36	2.20	10.06	6.16	0.95	2.93	1.11	15.76	0.58	12.56	2.99	1.31	21.66
Distal	0.20	0.15	3.03	3.84	10.28	0.08	0.75	1.50	2.42	11.07	6.77	1.05	3.23	1.23	17.33	0.64	13.81	3.29	1.44	23.83
Occlusal	11.75	9.00	181.75	230.25	617.00	5.00	45.00	90.00	145.00	664.00	406.25	62.75	193.50	73.50	1040.00	38.25	828.75	197.25	56.50	1429.75
Cuspids																				
Upper 6, 11																				
Lingual	0.32	0.24	4.91	6.22	16.68	0.14	1.22	2.43	3.92	17.95	10.98	1.70	5.23	1.99	28.11	1.03	22.40	5.33	2.34	38.64
Buccal	0.29	0.23	4.54	5.76	15.43	0.13	1.13	2.25	3.63	16.60	10.16	1.57	4.84	1.84	26.00	0.96	20.72	4.93	2.16	35.74
Mesial	0.08	0.06	1.25	1.59	4.26	0.03	0.31	0.62	1.00	4.58	2.80	0.43	1.33	0.51	7.17	0.26	5.72	1.36	0.60	9.86
Distal	0.05	0.04	0.83	1.05	2.81	0.02	0.21	0.41	0.66	3.03	1.85	0.29	0.88	0.33	4.74	0.17	3.78	0.90	0.39	6.51
Occlusal	0.52	0.40	8.08	10.23	27.42	0.22	2.00	4.00	6.44	29.51	18.06	2.79	8.60	3.27	46.22	1.70	36.83	8.77	3.84	63.54
Lower 22, 27																				
Lingual	3.36	2.57	51.93	65.79	176.29	1.43	12.86	25.71	41.43	189.71	116.07	17.93	55.29	21.00	297.14	10.93	236.79	56.36	24.71	408.50
Buccal	0.30	0.23	4.60	5.83	15.62	0.13	1.14	2.28	3.67	16.81	10.28	1.59	4.90	1.86	26.33	0.97	20.98	4.99	2.19	36.20
Mesial	0.35	0.27	5.47	6.92	18.56	0.15	1.35	2.71	4.36	19.97	12.22	1.89	5.82	2.21	31.28	1.15	24.92	5.93	2.60	43.00
Distal	0.30	0.23	4.69	5.94	15.92	0.13	1.16	2.32	3.74	17.14	10.48	1.62	4.99	1.90	26.84	0.99	21.39	5.09	2.23	36.90
Occlusal	0.16	0.12	24.23	30.70	8.23	0.67	6.00	12.00	19.33	88.53	54.17	8.37	25.80	9.80	138.67	5.10	110.50	26.30	11.53	190.63
1st Bicuspids																				
Upper 5, 12																				
Lingual	0.94	0.72	14.54	18.42	49.36	0.40	3.60	7.20	11.60	53.12	32.50	5.02	15.48	5.88	83.20	3.06	66.30	15.78	6.92	114.38
Buccal	0.50	0.38	7.73	9.80	26.26	0.21	1.91	3.83	6.17	28.26	17.29	2.67	8.23	3.13	44.26	1.63	35.27	8.39	3.68	60.84
Mesial	0.10	0.08	1.58	2.00	5.37	0.04	0.39	0.78	1.26	5.77	3.53	0.55	1.68	0.64	9.04	0.33	7.21	1.72	0.75	12.43
Distal	0.05	0.04	0.71	0.90	2.42	0.02	0.18	0.35	0.57	2.61	1.59	0.25	0.76	0.29	4.08	0.15	3.25	0.77	0.34	5.61
Occlusal	0.02	0.02	0.34	0.43	1.15	0.01	0.08	0.17	0.27	1.24	0.76	0.12	0.36	0.14	1.94	0.07	1.54	0.37	0.16	2.66
Lower 21, 28																				
Lingual	2.61	2.00	40.39	51.17	137.11	1.11	10.00	20.00	32.22	147.56	90.28	13.94	43.00	16.33	231.11	8.50	184.17	43.83	19.22	317.72
Buccal	0.36	0.27	5.51	6.98	18.70	0.15	1.36	2.73	4.39	20.12	12.31	1.90	5.86	2.23	31.52	1.16	25.11	5.98	2.62	43.33
Mesial	0.29	0.22	4.43	5.62	15.05	0.12	1.10	2.20	3.54	16.20	9.91	1.53	4.72	1.79	25.37	0.93	20.21	4.81	2.11	34.87
Distal	0.16	0.12	2.49	3.15	8.45	0.07	0.61	1.23	1.99	9.10	5.57	0.86	2.65	1.01	14.25	0.52	11.35	2.70	1.18	19.59
Occlusal	0.05	0.04	0.75	0.95	2.56	0.02	0.19	0.37	0.60	2.75	1.68	0.26	0.80	0.30	4.31	0.16	3.44	0.82	0.36	5.93

TABLE 3 (in eight parts)

Cavities per 100 erupted teeth. Ratio of numbers of each kind of cavity in each pair of teeth to each other kind of cavity in each other pair of teeth

Eighth part

	SECOND MOLARS										THIRD MOLARS									
	Upper 2, 15					Lower 18, 31					Upper 1, 16					Lower 17, 32				
	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O	L	B	M	D	O
Centrals																				
Upper 8, 9																				
Lingual	2.52	2.15	0.74	0.89	18.31	0.36	6.99	1.22	0.44	26.71	0.50	2.59	0.33	0.50	15.03	0.22	4.31	0.52	0.25	19.74
Buccal	5.18	4.42	1.51	1.83	37.59	0.74	14.35	2.51	0.90	54.83	1.03	5.32	0.67	1.03	39.07	0.46	8.85	1.07	0.52	40.53
Mesial	0.25	0.33	0.07	0.09	1.85	0.04	0.71	0.12	0.04	2.69	0.05	0.26	0.03	0.05	1.92	0 02	0.43	0.05	0.03	0.99
Distal	0.31	0.27	0.09	0.11	2.28	0.05	0.87	0.15	0.05	3.33	0.06	0.32	0.04	0.06	2.37	0.03	0.54	0.06	0.03	2.46
Occlusal	18.13	15.47	5.29	6.42	131.55	2.61	50.21	8.79	3.16	191.89	3.61	18.61	2.34	3.61	136.74	1.61	30.97	3.74	1.82	141.84
Lower 24, 25																				
Lingual	344.50	294.00	100.50	122.00	2499.50	49.50	954.00	167.00	60.00	3646.00	68.50	353.50	44.50	68.50	2598.00	30.50	588.50	71.00	34.50	2695.00
Buccal	62.64	53.45	18.27	22.18	454.45	9.00	173.45	30.36	10.91	662.91	12.45	64.27	8.09	12.45	472.36	0.55	10.70	12.91	6.27	490.00
Mesial	2.08	1.77	0.61	0.73	15.06	0.30	5.75	1.01	0.36	21.96	0.41	2.13	0.27	0.41	15.65	0.18	3.55	0.43	0.21	16.23
Distal	2.38	2.03	0.69	0.84	17.24	0.34	6.58	1.15	0.41	25.14	0.47	2.44	0.31	0.47	17.92	0.21	4.06	0.49	0.24	18.59
Occlusal	86.13	73.50	25.13	30.50	624.88	12.38	238.50	41.75	15.00	911.50	17.13	88.38	11.13	17.13	649.50	7.63	147.13	17.75	8.63	673.75
Laterals																				
Upper 7, 10																				
Lingual	0.69	0.59	0.20	0.25	5.02	0.10	1.92	0.34	0.12	7.33	0.14	0.71	0.09	0.14	5.22	0.06	1.18	0.14	0.07	5.42
Buccal	4.96	4.23	1.45	1.76	35.96	0.71	13.73	2.40	0.86	52.46	0.99	5.09	0.64	0.99	37.38	0.44	8.47	1.02	0.50	38.78
Mesial	0.25	0.22	0.07	0.09	1.84	0.04	0.70	0.12	0.04	2.68	0.05	0.26	0.03	0.05	1.91	0.02	0.43	0.05	0.03	1.98
Distal	0.65	0.56	0.19	0.23	4.75	0.09	1.81	0.32	0.11	6.93	0.13	0.67	0.08	0.13	4.94	0.06	1.12	0.13	0.07	5.12
Occlusal	16.02	13.67	4.67	5.67	116.26	2.30	44.37	7.77	2.79	169.58	3.19	16.44	2.07	3.19	120.84	1.42	27.37	3.30	1.60	125.35
Lower 23, 26																				
Lingual	68.90	58.80	20.10	24.40	499.90	9.90	190.80	33.40	12.00	729.20	13.70	70.70	8.90	13.70	519.60	6.10	117.70	14.20	6.90	539.00
Buccal	23.76	20.28	6.93	8.41	172.38	3.41	65.79	11.52	4.14	251.45	4.72	2.44	3.07	4.72	179.17	2.10	40.59	4.90	2.38	185.86
Mesial	2.61	2.23	0.76	0.92	18.94	0.38	7.23	1.27	0.45	27.62	0.52	2.68	0.34	0.52	19.68	0.23	4.46	0.54	0.26	20.42
Distal	2.87	2.45	0.84	1.02	20.83	0.41	7.95	1.39	0.50	30.38	0.57	2.95	0.37	0.57	21.65	0.25	4.90	0.59	0.29	22.46
Occlusal	172.25	147.00	50.25	61.00	1249.75	24.75	477.00	83.50	30.00	1823.00	34.25	176.75	22.25	34.25	1299.00	15.25	294.25	35.50	17.25	1347.50
Cuspids																				
Upper 6, 11																				
Lingual	4.66	3.97	1.36	1.65	33.78	0.67	12.89	2.26	0.81	49.27	0.93	4.78	0.60	0.93	35.11	0.41	7.95	0.96	0.47	36.42
Buccal	4.31	3.68	1.26	1.53	31.24	0.62	11.93	2.09	0.75	45.58	0.86	4.42	0.56	0.86	32.48	0.38	7.36	0.89	0.43	33.69
Mesial	1.19	1.01	0.35	0.42	8.62	0.17	3.29	0.58	0.21	12.57	0.24	1.22	0.15	0.24	8.96	0.11	2.03	0.24	0.12	9.29
Distal	0.78	0.67	0.23	0.28	5.69	0.11	2.17	0.38	0.14	8.31	0.16	0.81	0.10	0.16	5.92	0.07	1.34	0.16	0.08	6.14
Occlusal	7.66	6.53	2.23	2.71	55.54	1.10	21.20	3.71	1.33	81.02	1.52	7.86	0.10	1.52	57.73	0.68	1.31	1.58	0.77	59.89
Lower 22, 27																				
Lingual	49.21	42.00	14.36	17.43	357.07	7.07	136.29	23.86	8.57	520.86	9.79	50.50	6.36	9.79	371.14	4.36	84.07	10.14	4.93	385.00
Buccal	4.36	3.72	1.27	1.54	31.64	0.63	12.08	2.11	0.76	46.15	0.87	4.47	0.56	0.87	32.89	0.39	7.45	0.90	0.44	34.11
Mesial	5.18	4.42	1.51	1.83	37.59	0.74	14.35	2.51	0.90	54.83	1.03	5.32	0.67	1.03	39.07	0.46	8.85	1.07	0.52	40.53
Distal	4.45	3.79	1.30	1.57	32.25	0.64	12.31	2.15	0.77	47.05	0.88	4.56	0.57	0.88	33.52	0.39	7.59	0.92	0.45	34.77
Occlusal	22.97	19.60	6.70	8.13	166.63	3.30	63.60	11.13	4.00	243.07	4.57	23.57	2.97	4.57	173.20	2.03	39.23	4.73	2.30	179.67
1st Bicuspids																				
Upper 5, 12																				
Lingual	13.78	11.76	4.02	4.88	99.98	1.98	38.16	6.68	2.40	145.84	2.74	14.14	1.78	2.74	103.92	1.22	23.54	2.84	1.38	107.80
Buccal	7.41	6.32	2.16	2.62	53.75	1.06	20.52	3.59	1.29	78.41	1.46	7.52	0.95	1.46	55.28	0.65	12.52	1.51	0.73	57.34
Mesial	1.50	1.28	0.44	0.53	10.87	0.22	4.15	0.73	0.26	15.85	0.30	1.54	0.19	0.30	11.30	0.13	2.56	0.31	0.15	11.72
Distal	0.68	0.58	0.20	0.24	4.91	0.10	1.87	0.33	0.12	7.16	0.13	0.69	0.09	0.13	5.10	0.06	1.16	0.14	0.07	5.29
Occlusal	0.32	0.27	0.09	0.11	2.33	0.05	0.89	0.16	0.06	3.40	0.06	0.33	0.04	0.06	2.42	0.03	0.55	0.07	0.03	2.54
Lower 21, 28																				
Lingual	38.28	32.67	11.17	13.56	277.72	5.50	106.00	18.56	6.67	405.11	7.61	39.28	4.94	7.61	288.67	3.39	65.39	7.89	3.83	299.44
Buccal	5.22	4.45	1.52	1.85	37.87	0.75	14.45	2.53	0.91	55.24	1.04	5.36	0.67	1.04	39.36	0.46	8.92	1.08	0.52	40.83
Mesial	4.20	3.59	1.23	1.49	30.48	0.60	11.63	2.04	0.73	44.46	0.84	4.31	0.54	0.84	31.68	0.37	7.18	0.87	0.42	32.87
Distal	2.36	2.01	0.69	0.84	17.12	0.34	6.53	1.14	0.41	24.97	0.47	2.42	0.30	0.47	17.79	0.21	4.03	0.49	0.24	18.40
Occlusal	0.71	0.61	0.21	0.25	5.18	0.10	1.98	0.35	0.12	7.56	0.14	0.73	0.09	0.14	5.38	0.06	1.22	0.15	0.07	5.59

Figs. 21 and *22* also show the relative susceptibility of all the other tooth surfaces. No allowance has here been made for the difference in the size of the surfaces, as for example the contrast between the small occlusal surfaces of the incisors, and the broad surfaces of the

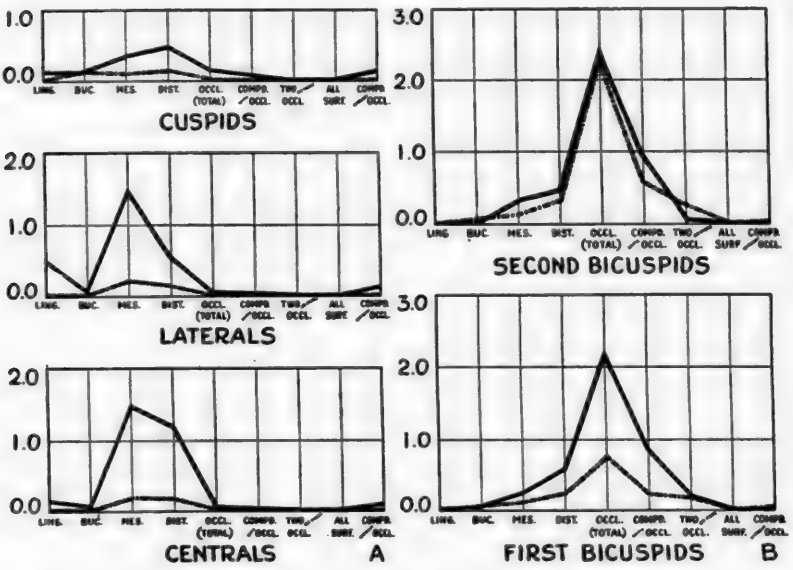

FIG. 21. Graphs showing per cent of total cavities in specified tooth surfaces, proportionate allowance having been made for missing and badly decayed teeth. Examination of 2943 males and females under 25 years of age. Unbroken lines—*upper* teeth; broken lines—*lower* teeth.

Graph A.—Centrals, laterals, and cuspids. *Graph B.*—First bicuspids and second bicuspids. At the bottom of the units in each graph, the abbreviations for the last four dental terms signify, respectively, "compound *with* occlusal," "two or more occlusal," "all surfaces," and "compound *without* occlusal." See *fig. 22* for related data.

molars; but such an allowance is very easily made mentally and in fact, almost instinctively, in reading the chart.

Another way of summarizing certain of the facts brought out in this statistical study is seen in *Table 3*, which, in each of its eight parts, is to be read somewhat after the manner of a multiplication table, except that it gives quotients or ratios instead of products. For

example, if one wishes to ascertain the ratio of the susceptibility to decay of the distal surface of the upper laterals to the occlusal surface of the upper first molar, he looks for the column headed "Laterals,

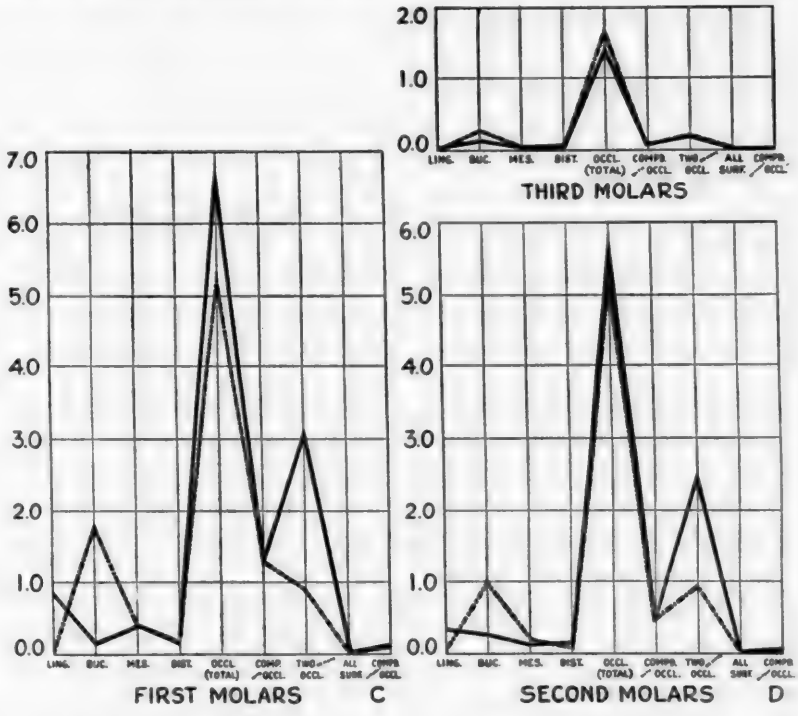

FIG. 22. Graphs showing per cent of total cavities in specified tooth surfaces, proportionate allowance having been made for missing and badly decayed teeth. Examination of 2943 males and females under 25 years of age. Unbroken lines—*upper* teeth; broken lines—*lower* teeth.

Graph C.—First molars. *Graph D.*—Second molars and third molars. At the bottom of the units in each graph, the abbreviations for the last four dental terms signify, respectively, "compound *with* occlusal," "two or more occlusal," "all surfaces," and compound *without* occlusal." See *fig. 21* for related data.

Upper, Distal," and reads, in the line, "First Molars, Upper, Occlusal," the figure, 0.25. This means that the distal surface of the upper laterals is only one-fourth, or 25 per cent, as susceptible to decay as the occlusal surface of the upper first molars. (See *Table 3*, Third part.)

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